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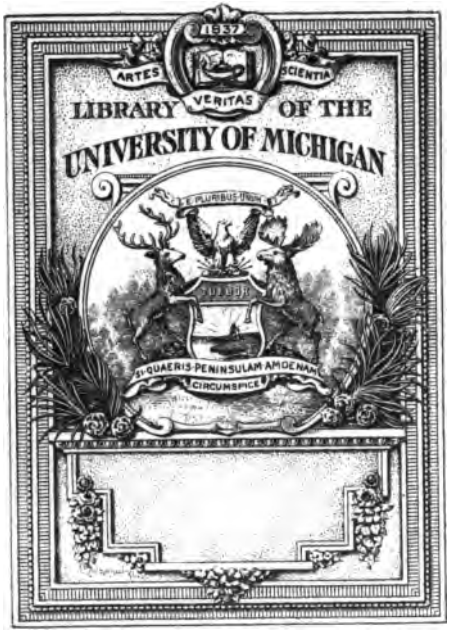
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THE ROYAL

MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON

VOLUME THE SEVENTY-SIXTH

(SECOND SERIES VOLUME THE FIFTY-EIGHTH)

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ELECTED

1805. WILLIAM SAUNDERS, M.D.
1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, Bart., M.D., G.C.H.
1813. SIR GILBERT BLANE, Bart., M.D.
1815. HENRY CLINE
1817. WILLIAM BABINGTON, M.D.
1819. SIR ASTLEY PASTON COOPER, Bart., K.C.H., D.C.L.
1821. JOHN COOKE, M.D.
1823. JOHN ABERNETHY
1825. GEORGE BIRKBECK, M.D.
1827. BENJAMIN TRAVERS
1829. PETER MARK ROGET, M.D.
1831. SIR WILLIAM LAWRENCE, Bart.
1833. JOHN ELLIOTSON, M.D. (First President of the Royal Medical and Chirurgical Society, after its Incorporation, 1834)*
1835. HENRY EARLE
1837. RICHARD BRIGHT, M.D., D.C.L.
1839. SIR BENJAMIN COLLINS BRODIE, Bart., D.C.L.
1841. ROBERT WILLIAMS, M.D.
1843. EDWARD STANLEY
1845. WILLIAM FREDERICK CHAMBERS, M.D., K.C.H.
1847. JAMES MONCRIEFF ARNOTT
1849. THOMAS ADDISON, M.D.
1851. JOSEPH HODGSON
1853. JAMES COPLAND, M.D.
1855. CÆSAR HENRY HAWKINS
1857. SIR CHARLES LOCOCK, Bart., M.D.
1859. FREDERIC CARPENTER SKEY
1861. BENJAMIN GUY BABINGTON, M.D.
1863. RICHARD PARTRIDGE
1865. SIR JAMES ALDERSON, M.D., D.C.L.
1867. SAMUEL SOLLY
1869. SIR GEORGE BURROWS, Bart., M.D., D.C.L.
1871. THOMAS BLIZARD CURLING
1873. CHARLES JAMES BLASIUS WILLIAMS, M.D.
1875. SIR JAMES PAGET, Bart., D.C.L., LL.D.
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1879. JOHN ERIC ERICHSEN
1881. ANDREW WHYTE BARCLAY, M.D.
1882. JOHN MARSHALL
1884. SIR GEORGE JOHNSON, M.D.
1886. GEORGE DAVID POLLOCK
1888. SIR EDWARD HENRY SIEVEKING, M.D., LL.D.
1890. TIMOTHY HOLMES
1892. SIR ANDREW CLARK, Bart., M.D., LL.D., F.R.S.

* From 1805 to 1834 the Society was known as the Medico-Chirurgical Society of London.

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1887 FLOWER, SIR WILLIAM HENRY, K.C.B., LL.D., F.R.S., Director of the Natural History Department, British Museum, Cromwell road.

1887 FOSTER, MICHAEL, LL.D., F.R.S., Professor of Physiology in the University of Cambridge.

1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Corresponding Member of the Academy of Sciences of France; The Yews, Reigate Hill, Reigate.

1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L., LL.D., F.R.S., Corresponding Member of the Academy of Sciences of France; The Camp, Sunningdale.

1868 HUXLEY, the Right Hon. THOMAS HENRY, D.C.L., LL.D., F.R.S., Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, and Dresden.

1878 LUBBOCK, the Right Hon. SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Farnborough, Kent, R.S.O.

1873 STOKES, SIR GEORGE GABRIEL, Bart., M.A., D.C.L., LL.D., F.R.S., M.P., Lucasian Professor of Mathematics in the University of Cambridge; Lensfield Cottage, Cambridge.

Elected

1887 **Turner, Sir William, D.C.L., LL.D., F.R.S., Professor of Anatomy in the University of Edinburgh; 6, Eton Terrace, Edinburgh.**

1868 **Tyndall, John, D.C.L., LL.D., F.R.S., Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, and Geneva; Hindhead House, Hindhead, Surrey.**

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1878 BACCELLI, GUIDO, M.D., Professor of Medicine at Rome.

1887 BILLINGS, JOHN S., M.D., D.C.L. Oxon., Surgeon U.S.Army; Librarian, Surgeon-General's Office, Washington.

1876 BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna; 20, Alger Strasse, Vienna.

1883 DUBOIS REYMOND, EMIL, M.D., Professor of Physiology, Berlin; N. W. Neue Wilhelmstrasse 15, Berlin.

1887 VON ESMARCH, FRIEDRICH, M.D., Professor of Surgery in the University of Kiel.

1866 HANNOVER, ADOLPH, M.D., Copenhagen.

1873 VON HELMHOLTZ, HERMANN LUDWIG FERDINAND, Professor of Physics and Physiological Optics; Berlin.

1868 KÖLLIKER, ALBERT, Professor of Anatomy in the University of Würzburg.

1868 LARREY, HIPPOLYTE BARON, Member of the Institute of France; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1883 PASTEUR, LOUIS, LL.D., Member of the Institute of France.

1878 VON SCANZONI, FRIEDREICH WILHELM, Royal Bavarian Privy Councillor; Professor of Midwifery in the University of Würzburg.

Elected

1856 VON VIRCHOW, RUDOLPH, M.D., LL.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of France; 10, Schellingstrasse, Berlin.

FELLOWS

OF THE

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OF LONDON.

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T.—Treasurer.	S.—Hon. Secretary.
L.—Hon. Librarian.	C.—Member of Council.
Sci. Com.—A Scientific Committee.	*Lib. Com.*—Library Committee.
Ho. Com.—House Committee.	*Bldg. Com.*—Building Committee.

The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Referee, Sci. Com.*, and *Lib. Com.*, *Bldg. Com.*, *Ho. Com.*, with the dates of office, are attached to the names of those who have served as Referees of papers and on the Committees of the Society.

OCTOBER, 1893.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

†1877 ABERCROMBIE, JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. *Trans.* 1.

1885 ABRAHAM, PHINEAS S., M.A., M.D., Lecturer on Physiology and Histology at the Westminster Hospital; 2, Henrietta street, Cavendish square.

*1851 ACLAND, SIR HENRY W., Bart., K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine in the University of Oxford; Oxford.

Elected

1885 ACLAND, THEODORE DYKE, M.D., Assistant Physician to St. Thomas's Hospital and to the Hospital for Consumption and Diseases of the Chest, Brompton; 74, Brook street, Grosvenor square, W.

†1852 ADAMS, WILLIAM, Consulting Surgeon to the Great Northern Central Hospital, the National Hospital for the Paralysed and Epileptic, and the National Orthopædic Hospital; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 12, Ladbroke terrace, Notting hill.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, A.M., M.D., LL.D.Glasgow, F.R.S., Regius Professor of Physic, Univ. Camb.; Consulting Physician to the Leeds General Infirmary; St. Rhadegund's, Cambridge. *Trans.* 8.

1879 ALLCHIN, WILLIAM HENRY, M.D., F.R.S. Ed., Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; 5, Chandos street, Cavendish square.

1890 ALLINGHAM, HERBERT WILLIAM, 25, Grosvenor street, Grosvenor square.

1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 48, Harley street, Cavendish square. *Trans.* 2.

1884 ANDERSON, ALEXANDER RICHARD, Surgeon to the General Hospital, 5, East Circus Street, Nottingham. *Trans.* 1.

1888 ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich; 9, Harley street, Cavendish square.

1890 ANDERSON, WILLIAM, Surgeon to St. Thomas's Hospital; Professor of Anatomy to the Royal Academy of Arts; 2, Harley street, Cavendish square.

Elected

1862 ANDREW, JAMES, M.D., Sunnycote, Emery Down, Lyndhurst, Hants. S. 1878-9. C. 1881-2. V.P. 1888. *Trans.* 1.

1891 ANDREWES, FREDERICK WILLIAM, M.B., 35, Welbeck street, Cavendish square.

1891 ANDREWS, LAUNCELOT, M.B., 22, Cheyne gardens, Manor street, Chelsea.

*1880 APPLETON, HENRY, M.D., 6, Southbourne terrace, Bournemouth.

1888 ARKLE, CHARLES, M.B., 66, Wimpole street, Cavendish square.

1893 BAILEY, ROBERT COZENS, M.S., M.B., 2, Museum Chambers, Bury street, Bloomsbury.

1891 BAKER, CHARLES ERNEST, M.B., Marlborough road, St. Albans.

*1873 BAKER, J. WRIGHT, Consulting Surgeon to the Derbyshire General Infirmary [care of Dr. Benthall, 101, Friar gate, Derby].

1865 BAKER, WILLIAM MORRANT, Consulting Surgeon to the Evelina Hospital for Sick Children; 39, Woburn square. C. 1878-9. V.P. 1889. *Referee*, 1886-8. *Lib. Com.* 1876-7. *Trans.* 7.

1891 BALGARNIE, WILFRED, M.B.

1887 BALL, JAMES BARRY, M.D., 12, Upper Wimpole street, Cavendish square.

1885 BALLANCE, CHARLES ALFRED, M.S., Assistant Surgeon to St. Thomas's Hospital and to the Hospital for Sick Children, Great Ormond street; Surgeon to the National Hospital for the Paralysed and Epileptic, Queen square; 106, Harley street, Cavendish square. *Trans.* 1.

†1848 BALLARD, EDWARD, M.D., F.R.S., late Inspector, Medical Department, Local Government Board; 20, Curzon park, Chester. C. 1872. V.P. 1875-6. *Sci. Com.* 1889—. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

Elected

*1866 BANKS, SIR JOHN, M.D., LL.D., D.Sc., K.C.B., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to Sir Patrick Dunn's and City of Dublin Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Royal University in Ireland; 45, Merrion square, Dublin.

1886 BANKS, WILLIAM MITCHELL, M.D., Surgeon to the Liverpool Royal Infirmary; 28, Rodney street, Liverpool.

1879 BARKER, ARTHUR EDWARD JAMES, Professor of Clinical Surgery at University College, and Surgeon to University College Hospital, London; 87, Harley street, Cavendish square. *Trans.* 7.

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service.

1876 BARLOW, THOMAS, M.D., B.S., *Trustee for Debenture-holders*, Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond street; 10, Wimpole street, Cavendish square. C. 1892. *Trans.* 2.

*1881 BARNES, HENRY, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

†1861 BARNES, ROBERT, M.D., Liss, Hants. C. 1877-8. V.P. 1889–90. *Referee*, 1867-76, 1891—. *Lib. Com.* 1869-73. *Sci. Com.* 1889—. *Trans.* 4.

1893 BARRETT, HOWARD, 49, Gordon square.

1880 BARROW, A. BOYCE, Assistant Surgeon to King's College Hospital and to the Westminster Hospital; 37, Wimpole street, Cavendish square.

1840 BARROW, BENJAMIN, Consulting Surgeon to the Royal Isle of Wight Infirmary.

1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 12.

Elected

1868 Bastian, Henry Charlton, M.A., M.D., F.R.S., Professor of Medicine in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 8a, Manchester square. C. 1885. *Referee*, 1886—. *Trans.* 2.

1890 Bateman, William A. F., Bridge House, Richmond, Surrey.

1891 Batten, Frederick E., M.A., M.B., B.C.Cantab., 15, Airlie gardens, Campden hill, Kensington.

1875 Beach, Fletcher, M.B., Two Elms, Chislehurst road, Sidcup, Kent.

1883 Beale, Edwin Clifford, M.A., M.B., Physician to the City of London Hospital for Diseases of the Chest, and Physician to the Great Northern Central Hospital; 23, Upper Berkeley street.

1862 Beale, Lionel Smith, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.

*1860 Bealey, Adam, M.D., M.A., Filsham Lodge, Filsham road, St. Leonard's-on-Sea, Sussex.

1856 Beardsley, Amos, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1880 Beevor, Charles Edward, M.D., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, and the Great Northern Hospital; 33, Harley street, Cavendish square. *Trans.* 1.

1880 Bennett, Alex. Hughes, M.D., Physician to the Westminster Hospital; 76, Wimpole street, Cavendish square. *Referee*, 1892—. *Trans.* 1.

1883 Bennett, Storer, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to, and Lecturer on Dental Surgery and Pathology at, the Dental Hospital of London; 17, George street, Hanover square.

Elected

1877 BENNETT, WILLIAM HENRY, Surgeon to St. George's Hospital; 1, Chesterfield street, Mayfair. C. 1893—. *Referee*, 1892-93. *Trans.* 4.

1889 BENTLEY, ARTHUR J. M., M.D., Mena House, Pyramids, Cairo, Egypt.

1890 BERRY, DAVID ANDERSON, M.B., C.M., 117, Goldhawk Road.

†1845 BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1885 BERRY, JAMES, B.S., Demonstrator of Anatomy, St. Bartholomew's Hospital; Surgeon to, and Lecturer on Clinical Surgery at, the Royal Free Hospital; 60 Welbeck street, Cavendish square.

1872 BEVERLEY, MICHAEL, M.D., Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.

*1865 BICKERSTETH, EDWARD ROBERT, Consulting Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool. *Trans.* 1.

1892 BICKERSTETH, ROBERT ALEXANDER, M.A., M.B., Assistant Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool.

1878 BINDON, WILLIAM JOHN VEREKER, M.D., 48, St. Ann's street, Manchester.

†1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

†1849 BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.

†1851 BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

Elected

1881 Biss, Cecil Yates, M.D., Senior Assistant Physician to, and Lecturer on Pharmacology and Therapeutics at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 135, Harley street, Cavendish square. *Trans.* 2.

1865 Blanchet, Hilarion, Examiner to the College of Physicians and Surgeons, Lower Canada; 35, Conillard street, Quebec, Canada.

1865 Blandford, George Fielding, M.D., late Lecturer on Psychological Medicine at St. George's Hospital; 48, Wimpole street, Cavendish square. C. 1883-4.

1891 Bokenham, Thomas Jessopp, 9, Upper Wimpole street, Cavendish square.

†1846 Bostock, John Ashton, C.B., *Hon. Treasurer;* Hon. Surgeon to H.M. the Queen; Deputy Surgeon-General; 73, Onslow gardens, Brompton. C. 1861-2. V.P. 1870-71. T. 1888—. *Sci. Com.* 1867.

1890 Bostock, R. Ashton, Surgeon, Scots Guards, 73, Onslow gardens, Brompton.

1869 Bourne, Walter, M.D. (Travelling).

1882 Bowlby, Anthony Alfred, Assistant Surgeon to St. Bartholomew's Hospital; 24, Manchester square. *Trans.* 4.

*1870 Bowles, Robert Leamon, M.D., 16, Upper Brook street, Grosvenor square. *Trans.* 1.

1886 Boxall, Robert, M.D., Assistant Obstetric Physician to, and Lecturer on Practical Midwifery at, the Middlesex Hospital; 29, Weymouth street, Portland place.

1884 Boyd, Stanley, M.B., Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Surgeon to the Paddington Green Children's Hospital; 134, Harley street, Cavendish square. *Trans.* 1.

1890 Bradford, John Rose, M.D., D.Sc., Assistant Physician to University College Hospital; 52, Upper Berkeley street, Portman square.

Elected

1874 BRADSHAW, A. F., C.B., Surgeon Major-General, Principal Medical Officer, H.M.'s Forces in India; Simla, India. [Agents: Holt & Co., 17, Whitehall place.]

1883 BRADSHAW, JAMES DIXON, M.B., 30, George street, Hanover square.

*1867 BRETT, ALFRED T., M.D., Watford House, Watford, Herts.

1876 BRIDGES, ROBERT, M.B., Manor House, Yattendon, Newbury, Berks.

1867 BRIDGEWATER, THOMAS, M.B., LL.D., Harrow-on-the-Hill, Middlesex.

1890 BRINTON, ROLAND DANVERS, M.D., 8, Queen's Gate terrace.

1868 BROADBENT, SIR WILLIAM HENRY, Bart., M.D., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; Physician in Ordinary to H.R.H. the Prince of Wales; 84, Brook street, Grosvenor square. C. 1885. *Referee*, 1881-4, 1891—. *Trans.* 5.

†1851 BRODHURST, BERNARD EDWARD, Surgeon to the Royal Orthopædic Hospital and to the Royal Hospital for Incurables; Consulting Surgeon, Belgrave Hospital for Children; Corresponding Member Société de Chirurgie, Paris, and of the Academy of Sciences, Rome; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.

1891 BRODIE, CHARLES GORDON, Senior Demonstrator of Anatomy, Middlesex Hospital Medical School; Assistant Surgeon, North-West London Hospital; 30, Harley street, Cavendish square.

1892 BRONNER, ADOLPH, M.D., 33, Manor row, Bradford.

1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., LL.D., F.R.S., Member of the Institut de France (Academy of Sciences); Professor of Medicine at the College of France. *Sci. Com.* 1862.

Elected

1888 BROWNE, HENRY LANGLEY, Moor House, West Bromwich.

1878 BROWNE, SIR JAMES CRICHTON, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 61, Carlisle place Mansions, Victoria street.

1880 BROWNE, JAMES WILLIAM, M.B., 7, Norland place, Holland Park.

1881 BROWNE, JOHN WALTON, M.D., Surgeon to the Belfast Royal Hospital; Surgeon to the Belfast Ophthalmic Hospital; 10, College square N., Belfast.

1881 BROWNE, OSWALD AUCHINLECK, M.A., M.B., Physician to the Royal Hospital for Diseases of the Chest; 43, Bedford square.

1874 BRUCE, JOHN MITCHELL, M.D., *Hon. Secretary;* Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; Physician to the Hospital for Consumption, Brompton; 70, Harley street. C. 1892. *Sci. Com.* 1889—. *Referee,* 1886-91. *Lib. Com.* 1888-91. *Trans.* 2.

1871 BRUNTON, THOMAS LAUDER, M.D., D.Sc., LL.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee,* 1880-87. *Lib. Com.* 1882-7. *Trans.* 1.

1860 BRYANT, THOMAS, Consulting Surgeon to Guy's Hospital; Member of the Société de Chirurgie, Paris; 65, Grosvenor street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee,* 1882-4. *Lib. Com.* 1868-71. *Trans.* 13. *Pro.* 1.

1864 BUCHANAN, SIR GEORGE, M.D., F.R.S., late Medical Officer of the Local Government Board; Member of the Senate of the University of London; 27, Woburn square.

1864 BUCKLE, FLEETWOOD, M.D.

1889 BULL, WILLIAM CHARLES, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 35, Clarges street, Piccadilly.

1881 BULLER, AUDLEY CECIL, M.D.

Elected

1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Surgeon to the Grosvenor Hospital for Women and Children; 76, Brook street, Grosvenor square.

1873 BUTLIN, HENRY TRENTHAM, Surgeon to St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887-8. *Referee*, 1893—. *Trans.* 4.

1871 BUTT, WILLIAM F.

1883 BUXTON, DUDLEY WILMOT, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Anæsthetist to the Hospital for Paralysis and Epilepsy, Queen's square, and to the London Dental Hospital; 82, Mortimer street, Cavendish square.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. *Referee*, 1887—.

*1851 CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.

1890 CAGNEY, JAMES, M.A., M.D., in charge of Electrical Department, St. Mary's Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 93, Wimpole street, Cavendish square. *Trans.* 1.

1885 CAHILL, JOHN, 12, Seville street, Lowndes square.

1893 CALEY, HENRY ALBERT, M.D., 19, Lower Seymour street, Portman square.

1887 CALVERT, JAMES, M.D., 36, Queen Anne street, Cavendish square.

1891 CAMPBELL, HENRY JOHNSTONE, M.D., Senior Demonstrator of Biology and Demonstrator of Physiology, Guy's Hospital; Assistant Physician, East London Children's Hospital; 54, Welbeck street.

1888 CARLESS, ALBERT, M.B., M.S., Assistant Surgeon to King's College Hospital; 10, Welbeck street.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.

Elected

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne street, Cavendish square. *Trans.* 1.

1888 CARTER, WILLIAM JEFFREYS BECHER, Aliwal North, Cape Colony.

1888 CAUTLEY, EDMUND, M.B., B.C., 15, Upper Brook street.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. C. 1887. *Lib. Com.* 1888—. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Consulting Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.

1884 CHAFFEY, WAYLAND CHARLES, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpellier road, Brighton.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.D., Physician Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; 42, Upper Brook street, Grosvenor square. *Referee*, 1891—. *Lib. Com.* 1885—. *Trans.* 7.

1859 CHANCE, FRANK, M.B., Burleigh House, Sydenham Hill.

1885 CHAPMAN, PAUL MORGAN, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.

1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; 2, Edinburgh Mansions, Victoria street, Westminster.

*1881 CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; Consulting Surgeon to the Bromsgrove Hospital; 22, Temple row, Birmingham. *Trans.* 3.

Elected

1868 CHEADLE, WALTER BUTLER, M.D., *Trustee;* Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890-91. *Sci. Com.* 1889—. *Bldg. Com.* 1889-92. *Referee*, 1885. *Trans.* 1.

1879 CHEYNE, WILLIAM WATSON, M.B., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; 75, Harley street, Cavendish square. *Lib. Com.* 1886-8, 1891—. *Trans.* 1.

1890 CHILDS, CHRISTOPHER, M.D., 2, Royal terrace, Weymouth.

*1873 CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Central Hospital; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee*, 1873-80.

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. V.P. 1892-3. *Referee*, 1874-81.

Died Nov. 6th, 1893.

1860 CLARK, SIR ANDREW, Bart., M.D., LL.D., F.R.S., *President*, *Trustee*, Consulting Physician to, and Emeritus Professor of Clinical Medicine at, the London Hospital; 16, Cavendish square. C. 1875. V.P. 1888. P. 1892-3.

1879 CLARK, ANDREW, Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.

1892 CLARK, JAMES CHARLES, Croft House, Margate road, Southsea.

1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Central London Ophthalmic Hospital; Surgeon and Ophthalmic Surgeon to the Miller Hospital; 41, Lee terrace, Blackheath, and 112, Harley street.

1890 CLARKE, JAMES JACKSON, M.B., Curator of the Museum and Pathologist, St. Mary's Hospital, Paddington.

†1848 CLARKE, JOHN, M.D. C. 1866.

Elected

1888 CLARKE, ROBERT HENRY, M.B., Westwood, Isle of Thanet, Kent.

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the West London Hospital; 46, Harley street, Cavendish square. *Trans.* 1.

†1879 CLUTTON, HENRY HUGH, M.A., M.B., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Surgeon to the Victoria Hospital for Children; 2, Portland place.

1857 COATES, CHARLES, M.D., Consulting Physician to the Bath Royal United Hospital; 10, Circus, Bath.

1888 COCK, FREDERICK WILLIAM, M.D., 1, Porchester Houses, Porchester square.

1868 COCKLE, JOHN, A.M., M.D., F.L.S., Consulting Physician to the Royal Free Hospital; 5, Suffolk place, Pall Mall. *Trans.* 2.

1893 COLE, ROBERT HENRY, M.B., Moorcroft, Hillingdon, Uxbridge.

1891 COOK, HERBERT GEORGE, M.B., B.S., 162, Cromwell road, South Kensington.

1865 COOPER, ALFRED, Consulting Surgeon to the West London Hospital; Senior Surgeon to St. Mark's Hospital, 9, Henrietta street, Cavendish square.

1868 CORNISH, WILLIAM ROBERT, C.I.E., late Surgeon-General to the Government of Madras; Hon. Physician to H.M. the Queen; 8, Cresswell gardens, The Boltons.

*1860 CORRY, THOMAS CHARLES STEUART, M.D., Ormeau terrace, and 1, Glenfield place, Belfast.

1889 COSENS, CHARLES HENRY, 49, Oxford terrace, Hyde Park.

1892 COTTERELL, EDWARD, 5, West Halkin street, Belgrave square.

1891 COUMBE, JOHN BATTEN, M.D., Wargrave, Henley-on-Thames.

†1860 COUPER, JOHN, Surgeon to the Royal London Ophthalmic Hospital, and Consulting Surgeon to the London Hospital; 80, Grosvenor street. C. 1876. *Referee*, 1882-3.

Elected

1877 COUPLAND, SIDNEY, M.D., Physician to, and Joint Lecturer on Practical Medicine at, the Middlesex Hospital; 16, Queen Anne street, Cavendish square. C. 1893—. *Referee*, 1892-3.

†1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.

1864 CRAWFORD, SIR THOMAS, K.C.B., M.D., M.Ch., LL.D., Hon. Surgeon to the Queen; Director-General, Army Medical Department (Retired); 5, St. John's Park, Blackheath. C. 1887.

[illegible] CROSSWELL, PEARSON R., Surgeon to the Merthyr General Hospital; Dowlais, Merthyr Tydvil.

1874 CRUISE, WILLIAM HARRISON, Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. C. 1890-91. *Trans.* 1.

1877 CULLERNE, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; late Physician to the East London Hospital for Children; 121, Harley street, Cavendish square. *Trans.* 3.

1858 CURNOW, JOHN, Consulting Surgeon to St. Thomas's Hospital; 6, Mansfield street, Cavendish square. C. 1884. V.P. 1890. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans* 2.

1878 CURTIS, FRANCIS RICHARDSON, M.B., Ophthalmic Surgeon to the Bristol Royal Infirmary, and Surgeon to the Bristol Eye Hospital; Worcester House, Clifton, Bristol.

1878 CUTLER, THOMAS HENRY RICKARD, 8, Campden Hill road, Kensington.

1866 DAVIES-COLLEY, CHARLES JAMES, M.D., Obstetric Physician and Lecturer on Midwifery at St. Thomas's Hospital; 40, Brook street, Grosvenor square.

1879 DAWBARN, ALBERT HERBERT, Aural Surgeon to St. Bartholomew's Hospital, and National Hospital for the Paralysed and Epileptic; 17, Queen Anne street, Cavendish square.

Elected

1873 CURNOW, JOHN, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital; Senior Physician to the Seamen's Hospital; 3, George street, Hanover square. *Referee*, 1884—.

1886 DAKIN, WILLIAM RADFORD, M.D., Obstetric Physician to St. George's Hospital; 57, Welbeck street, Cavendish square.

1872 DALBY, SIR WILLIAM BARTLETT, M.B., Consulting Aural Surgeon to St. George's Hospital; 18, Savile row. *Trans.* 3.

1884 DALLAWAY, DENNIS, 5, Duchess street, Portland place.

1891 DALTON, NORMAN, M.D., 4, Mansfield street, Cavendish square.

1879 DARWIN, FRANCIS, M.B., F.R.S., Wychfield, Huntingdon road, Cambridge.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Royal Infirmary; 2, Gambier terrace, Liverpool.

1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 36, Harley street, Cavendish square. C. 1892-3. *Referee*, 1890-91. *Trans.* 2.

1893 DAVIS, GEORGE WILLIAM, M.D., B.S., Sunningdale, Sidcup, Kent.

1878 DAVY, RICHARD, Consulting Surgeon to the Westminster Hospital; Burstone House, Bow, North Devon. *Trans.* 1.

*1882 DAWSON, YELVERTON, M.D., Heathlands, Southbourne-on-Sea, Hants.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1889 DEAN, HENRY PERCY, M.B., M.S., 84, Wimpole street, Cavendish square.

1889 DELÉPINE, SHERIDAN, B.S., M.B., Professor of Pathology, Owens College, Manchester. *Trans.* 1.

Elected

1878 DENT, CLINTON THOMAS, Assistant Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. C. 1890. *Bldg. Com.* 1890-2. *Referee*, 1892—. *Trans.* 4.

†1859 DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. C. 1874-5. V.P. 1887. *Referee*, 1869-73. 1882-6. *Sci. Com.* 1867, 1879, 1889—. *Trans.* 14.

†1891 DICKINSON, WILLIAM LEE, M.B., 9, Chesterfield street, Mayfair.

†1839 DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

1889 DODD, HENRY WORK, Assistant Surgeon to the Royal Free Hospital, and to the Royal Westminster Ophthalmic Hospital; Ophthalmic Surgeon to the West-End Hospital for Nervous Diseases; 136, Harley street, Cavendish square.

1888 DONELAN, JAMES, M.B., M.C., 2, Upper Wimpole street, Cavendish square.

1879 DONKIN, HORATIO BRYAN, M.B.Oxon., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 108, Harley street, Cavendish square.

1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. C. 1893—. *Lib. Com.* 1891-3. *Trans.* 4.

1891 DOVE, PERCY W., "Carshalton," Stapleton Hall Road, N.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Consulting Physician to the London Hospital; 81, Harley street, Cavendish square. C. 1880. V.P. 1890-91. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

Elected

1884 DRAGE, LOVELL, M.A., M.B., B.S.Oxon., Burleigh Mead, Hatfield, Herts.

1879 DREWITT, F. G. DAWTREY, M.D., Physician to the West London Hospital and to the Victoria Hospital for Children; 2, Manchester square.

1885 DRUMMOND, DAVID, M.D., 7, Saville place, Newcastle-on-Tyne.

1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.

1865 DRYSDALE, CHARLES ROBERT, M.D., Senior Physician to the Metropolitan Hospital; 23, Sackville street, Piccadilly.

†1865 DUCKWORTH, SIR DYCE, M.D., LL.D., Hon. Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885—. *Trans.* 2.

1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place. C. 1893—.

1871 DUKE, BENJAMIN, Windmill House, Clapham Common.

*1871 DUKES, CLEMENT, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.

1867 DUKES, MAJOR CHARLES, M.D., 6, Wellesley Villa, Wellesley road, Croydon.

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.

*1889 DUNCAN, JOHN, M.D., St. Petersburg, Russia.

1884 DUNCAN, WILLIAM, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.

Elected

1887 DUNN, HUGH PERCY, Assistant Ophthalmic Surgeon and Pathologist at the West London Hospital; 39, Welbeck street, Cavendish square.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. V.P. 1887. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 DURHAM, FREDERIC, M.B., Senior Surgeon to the North-West London Hospital; late Surgical Registrar to Guy's Hospital; 82, Brook street, Grosvenor square.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1887 EASMON, JOHN FARRELL, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony; Accra, Gold Coast, West Africa.

1868 EASTES, GEORGE, M.B.Lond., 35, Gloucester place, Hyde Park. C. 1892-3.

1888 ECCLES, ARTHUR SYMONS, M.B., C.M., 23, Hertford street, Mayfair.

1891 EDDOWES, ALFRED, M.D., 25, Old Burlington street.

1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 2.

1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; 55, Harley street, Cavendish square.

1891 ELAM, GEORGE, M.B., 3, Upper Montague street, Russell square.

1887 ELLIOTT, JOHN, Whitefriars Lodge, Chester.

1848 ELLIS, GEORGE VINER, Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

Elected

*1854 ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1889 ELLISTON, WILLIAM ALFRED, M.D., Stoke Hall, Ipswich.

†1842 ERICHSEN, JOHN ERIC, LL.D., F.R.S., M.Ch., Surgeon Extraordinary to H.M. the Queen; President of, and Emeritus Professor of Surgery in, University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. P. 1879-80. *Referee*, 1866-8, 1884-89. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 EVE, FREDERIC S., Surgeon to the London Hospital; Surgeon to Out-Patients at the Evelina Hospital for Sick Children; 125, Harley street, Cavendish square. *Trans.* 2.

1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital; 33, Curzon street, Mayfair. *Sci. Com.* 1889—. *Trans.* 1.

*1875 FAGAN, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 19, Great Victoria street, Belfast.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., 59, Warrior square, St. Leonard's-on-Sea.

1862 FARQUHARSON, ROBERT, M.D., LL.D., M.P., Migvie Lodge, Porchester gardens, Hyde Park (Finzean, Aboyne, Aberdeenshire). *Lib. Com.* 1876-80.

1872 FAYRER, SIR JOSEPH, K.C.S.I., LL.D., M.D., F.R.S., Surgeon-General; Honorary Physician to H.M. the Queen, (Military) to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Physician to the Secretary of State for India in Council; President of the Medical Board at the India Office; 53, Wimpole street, Cavendish square. C. 1888. *Referee*, 1881-7.

1887 FEENY, MICHAEL HENRY, Les Avants, Montreux, Switzerland.

*1872 FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 25, North road, Durham.

Elected

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882—. *Trans.* 4.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Neuropathology in King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Referee*, 1891—. *Trans.* 2.

1889 FIELD, GEORGE P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital, and Dean of the Medical School; 34, Wimpole street, Cavendish square.

1879 FINLAY, DAVID WHITE, M.D., Professor of the Practice of Medicine in the University of Aberdeen; Physician to the Aberdeen Royal Infirmary; Consulting Physician to the Royal Hospital for Diseases of the Chest, London; 2, Queen's terrace, Aberdeen. *Referee*, 1891—. *Trans.* 2.

1866 FITZ-PATRICK, THOMAS, A.M., M.D., 30, Sussex gardens, Hyde Park.

1891 FLETCHER, HERBERT MORLEY, M.B., 98, Harley street, Cavendish square.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.

*1864 FOLKER, WILLIAM HENRY, Consulting and late Hon. Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1877 DE FONMARTIN, HENRY, M.D., 1, Anchor Gate terrace, Portsea, Hants.

1892 FORSBROOK, WILLIAM HENRY RUSSELL, M.D., 139, Buckingham Palace road.

1865 FOSTER, SIR BALTHAZAR WALTER, M.D., M.P., Emeritus Professor of Medicine at the Queen's College, Birmingham, and Consulting Physician to the Birmingham General Hospital; 55, Temple row, Birmingham, and 11, George street, Hanover square, London.

Elected

1892 FOSTER, MICHAEL GEORGE, M.A., M.B., Great Shelford, Cambridge.

1882 FOWLER, JAMES KINGSTON, M.A., M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

1887 FOX, RICHARD HINGSTON, M.D., Physician to St. Luke's Mission Dispensary; 23, Finsbury square.

1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician for Skin Diseases to the Westminster Hospital, and to the Skin Department of the Paddington Green Hospital for Children; late Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

1871 FRANK, PHILIP, M.D., Cannes, France.

*1884 FRANKS, KENDAL, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin; Surgeon in Ordinary to the Lord Lieutenant; 6, Fitzwilliam square, Dublin. *Trans.* 2.

*1889 FREEMAN, HENRY WILLIAM, 24, The Circus, Bath.

1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.

1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, Mayfair.

1876 FURNER, WILLOUGHBY, M.D., Surgeon to the Sussex County Hospital; 13, Brunswick square, Brighton.

*1864 GAIRDNER, WILLIAM TENNANT, M.D., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow. *Trans.* 1.

Elected

†1874 GALABIN, ALFRED LEWIS, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole st., Cavendish square. C. 1892. *Referee*, 1882-91. *Lib. Com.* 1883-4. *Trans.* 2.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 10, Upper Cheyne row, Chelsea.

1885 GAMGEE, ARTHUR, M.D., F.R.S., Davos, Switzerland.

1865 GANT, FREDERICK JAMES, Consulting Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. *Referee*, 1886—. *Lib. Com.* 1882-5. *Trans.* 3.

1867 GARLAND, EDWARD CHARLES, Yeovil, Somerset.

1867 GARLIKE, THOMAS W., Malvern Cottage, Churchfield road, Ealing.

†1854 GARROD, SIR ALFRED BARING, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

1886 GARROD, ARCHIBALD EDWARD, M.A., M.D., Assistant Physician to the West London Hospital; 9, Chandos street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 5.

1879 GARSTANG, THOMAS WALTER HARROPP, Headingley House, Knutsford, Cheshire.

*1889 GASKELL, WALTER HOLBROOK, M.D., F.R.S., Lecturer on Physiology, University of Cambridge; Petersfield House, Parkside, Cambridge.

1887 GAY, JOHN, 119, Upper Richmond road, Putney.

1866 GEE, SAMUEL JONES, M.D., *Hon. Librarian, Chairman of Trustees for Debenture-holders*, Physician to St. Bartholomew's Hospital; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887—. *Sci. Com.* 1879. *Bldg. Com.* 1889-92. *Referee*, 1885-7. *Lib. Com.* 1871-6. *Trans.* 1.

Elected

1885 GELL, HENRY WILLINGHAM, M.B., 43, Albion street, Hyde Park.

1878 GERVIS, HENRY, M.D., Consulting Obstetric Physician to St. Thomas's Hospital; Consulting Physician to the Royal Maternity Charity; 40, Harley street, Cavendish square. *Referee*, 1884—. *Trans.* 1.

1884 GIBBES, HENEAGE, M.D., Professor of Pathology in the University of Michigan; Ann Arbor, Michigan, U.S.A.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place.

1893 GILES, ARTHUR EDWARD, M.D., B.Sc., 57, Queen Anne street.

1877 GODLEE, RICKMAN JOHN, M.S., *Hon. Secretary*, Surgeon to University College Hospital, and Teacher of Operative Surgery in University College, London, and Surgeon to the Hospital for Consumption, Brompton; Consulting Surgeon to the North-Eastern Hospital for Children; 19, Wimpole street, Cavendish square. S. 1892-3. *Referee*, 1886-91. *Trans.* 8.

†1870 GODSON, CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.

1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Senior Assistant Surgeon and Lecturer on Physiology at Guy's Hospital; 12, Queen Anne street, Cavendish square. *Trans.* 1.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.

1883 GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place.

1889 GOODSALL, DAVID HENRY, Surgeon to the Metropolitan Hospital; Surgeon to St. Mark's Hospital; 17, Devonshire place, Upper Wimpole street.

Elected

*1890 GORDON, WILLIAM, M.B., Barnfield Lodge, Exeter.

1893. GORDON, WILLIAM, M.B., M.C., 80, Elvaston place, Queen's gate.

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; 10, Queen Anne street, Cavendish square. C. 1892-3. *Lib. Com.* 1891. *Trans.* 2.

1891 GOW, WILLIAM J., M.D., 13, Upper Wimpole street, Cavendish square.

1873 GOWERS, WILLIAM RICHARD, M.D., F.R.S., Consulting Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. C. 1891. *Referee* 1888-90. *Lib. Com.* 1884-6. *Trans.* 7.

†1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 82, Gloucester terrace, Hyde park.

1892 GRANT, J. DUNDAS, M.A., M.D., 8, Upper Wimpole street, Cavendish square.

1868 GREEN, T. HENRY, M.D., Physician to Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee,* 1882-5.

1889 GREENE, GEORGE EDWARD JOSEPH, Monte Vista, Ferns, County Wexford.

*1875 GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee,* 1881.

1882 GRESSWELL, DAN ASTLEY, M.A., M.D., D.P.H., Melbourne, Victoria.

1885 GRIFFITH, WALTER SPENCER ANDERSON, M.D., Assistant Physician-Accoucheur, St. Bartholomew's Hospital; University Lecturer and Examiner in Obstetrics, Cambridge; 114, Harley street, Cavendish square.

1889 GRIFFITHS, JOSEPH, M.A., M.D., C.M., Assistant to the Professor of Surgery in the University of Cambridge; 17, Fitzwilliam street, Cambridge.

Elected

1868 GRIGG, WILLIAM CHAPMAN, M.D., Obstetric Physician to the Out-patients at the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in Hospital; Joint Lecturer on Forensic Medicine at the Westminster Hospital Medical School; 27, Curzon street, Mayfair.

1852 GROVE, JOHN, Pitt House, 15, Johnstown street, Bath.

1889 GUBB, ALFRED S, M.D.Paris; 29, Gower street.

1883 GUNN, ROBERT MARCUS, M.A., M.B., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the Hospital for Sick Children, Great Ormond Street, and to the National Hospital for the Paralysed and Epileptic; Assistant Ophthalmic Surgeon to University College Hospital; 54, Queen Anne street, Cavendish square.

1890 GUTHRIE, LEONARD GEORGE, M.B., B.S., Physician to the Regent's Park Hospital for Paralysis; Assistant Physician to the North-West London Hospital; Assistant Physician and Pathologist to the Children's Hospital, Paddington Green; 24, Upper George street, Bryanston square.

1886 HABERSHON, SAMUEL HERBERT, M.D., 70, Brook street, Grosvenor square.

1885 HAIG, ALEXANDER, M.D., Physician to the Metropolitan Hospital, and to the Royal Hospital for Children and Women; 7, Brook street, Grosvenor square. *Trans.* 6.

1890 HALE, CHARLES DOUGLAS BOWDICH, M.D., 3, Sussex place, Hyde Park.

1881 HALL, FRANCIS DE HAVILLAND, M.D., Physician to Out-patients and to the Throat Department at the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square. *Referee*, 1893—.

1891 HAMER, WILLIAM HEATON, M.D., 73, Dartmouth Park Hill, Highgate.

1870 HAMILTON, ROBERT, Consulting Surgeon to the Royal Southern Hospital, Liverpool; Magheraybuoy, Portrush, Co. Antrim, Ireland.

Elected

1889 HANDFIELD-JONES, MONTAGU, M.D., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Mary's Hospital; Physician to the British Lying-in Hospital; 35, Cavendish square.

†1856 HARE, CHARLES JOHN, M.D., *Hon. Treasurer*, Consulting Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887—. *Bldg. Com.* 1889-92.

†1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. V.P. 1891-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3, 1866, 1889—. *Trans.* 1.

1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882—. *Sci. Com.* 1879. *Trans.* 10.

1892 HAROLD, JOHN, 91, Harley street, Cavendish square.

1880 HARRIS, VINCENT DORMER, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Demonstrator of Physiology at St. Bartholomew's Hospital; 31, Wimpole street, Cavendish square.

1870 HARRISON, REGINALD, 6, Lower Berkeley Street, Portman square. *Trans.* 1.

1892 HARSANT, WILLIAM HENRY, 16, Pembroke road, Clifton, Bristol.

1890 HAVILAND, FRANK PAPILLON, M.B., B.C., 57, Warrior square, St. Leonard's-on-Sea.

1870 HAWARD, J. WARRINGTON, *Trustee for Debenture-holders*, Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. S. 1888-91. *Lib. Com.* 1881-4. *Sci. Com.* 1889—. *Bldg. Com.* (*Sec.*) 1889-92. *Ho. Com.* 1892—. *Trans.* 2.

1885 HAWKINS, FRANCIS HENRY, M.B., 59, Wimpole street, Cavendish square.

Elected

1891 HAWKINS, HERBERT PENNELL, M.B., B.C., Assistant Physician to St. Thomas's Hospital; 38, Weymouth street, Portland place.

1875 HAYES, THOMAS CRAWFORD, M.A., M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital, and Lecturer in Practical Obstetrics in King's College; Physician for Diseases of Women to Royal Free Hospital; 17, Clarges street, Piccadilly.

1860 HAYWARD, HENRY HOWARD, Consulting Surgeon Dentist to St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

1891 HAYWARD, JOHN ARTHUR, M.B., Chestnuts, Teddington.

1861 HAYWARD, WILLIAM HENRY, Oxford road, Bromley, Lancashire.

†1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-8. *Trans.* 3.

1882 HENSLEY, PHILIP JOHN., M.D., Assistant Physician and Lecturer on Forensic Medicine to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Referee*, 1892—. *Trans.* 1.

1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.

1891 HERRING, HERBERT T., M.B., B.S., 50, Harley street Cavendish square.

1883 HERRINGHAM, WILMOT PARKER, M.D., Medical Registrar, St. Bartholomew's Hospital; 13, Upper Wimpole street, Cavendish square. *Trans.* 1.

1887 HEWITT, FREDERIC WILLIAM, M.D., Instructor in, and Lecturer on Anæsthetics at, the London Hospital; Anæsthetist at the Dental Hospital of London; 10, George street, Hanover square. *Trans.* 1.

Elected

1880 HICKS, CHARLES CYRIL, M.D., Wokingham, Berks.

1873 HIGGENS, CHARLES, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

1890 HILL, G. WILLIAM, M.D., B.Sc., 24, Wimpole street, Cavendish square.

†1843 HOLDEN, LUTHER, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.D., Assistant Physician to the Sussex County Hospital; 8, Cambridge road, Brighton.

†1856 HOLMES, TIMOTHY, M.A., Consulting Surgeon to St. George's Hospital; Corresponding Member of the "Société de Chirurgie," Paris; 18, Great Cumberland place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. P. 1890-92. *Bldg. Com. (Chairman)* 1889-92. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5, 1892—. *Ho. Com.* 1892—. *Trans.* 8.

†1846 HOLT, BARNARD WIGHT, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington Gardens. C. 1862-3. V.P. 1879-80.

†1846 HOLTHOUSE, CARSTEN, 1, Bath terrace, Richmond. C. 1863. *Referee*, 1870-6. *Lib. Com.* 1859-60.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., Senior Physician to the West London Hospital; 43, Green street, Park lane.

1883 HORSLEY, VICTOR ALEXANDER HADEN, F.R.S., Assistant Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; Professor of Pathology in University College, London; 25, Cavendish square. *Trans.* 1.

Elected

1865 HOWARD, BENJAMIN, M.D. [New York, U.S.] *Trans.* 1.

1881 HOWARD, HENRY, M.B., Medical Officer of Health, Williamstown, Melbourne, Victoria.

1892 HOWARD, R. J. BLISS, M.D., 31, Queen Anne street, Cavendish square.

1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. *Sci. Com.* 1879. *Referee*, 1887-89. *Trans.* 3.

1886 HUDSON, CHARLES ELLIOTT LEOPOLD BARTON, Surgical Registrar, Middlesex Hospital; 6, Chandos street, Cavendish square.

†1857 HULKE, JOHN WHITAKER, F.R.S., *Hon. Librarian*, Surgeon to the Middlesex Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879—. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 11.

1889 HUMPHERY, FRANCIS WILLIAM, M.A., M.B., 63, Prince's gate.

1855 HUMPHRY, SIR GEORGE MURRAY, M.D., D.Sc., LL.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 9.

1882 HUMPHRY, LAURENCE, M.D., 3, Trinity street, Cambridge.

1889 HUNTER, WILLIAM, M.D., Assistant Physician to the London Fever Hospital; 54, Harley street.

1873 HUNTER, SIR W. GUYER, M.D., K.C.M.G., Hon. Surgeon to H.M. the Queen; formerly Principal of, and Professor of Medicine in, Grant Medical College, and Vice-Chancellor of the University, Bombay; Surgeon-General (Retired) Bombay Army; Consulting Physician to Charing Cross Hospital; 21, Norfolk crescent, Hyde Park.

1849 HUSSEY, EDWARD LAW, Coroner of the City of Oxford; 24, Winchester road, Oxford. *Trans.* 1.

Elected

†1856 HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee*, 1876-81, 1883—. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.

1888 HUTCHINSON, JONATHAN, Jun., Assistant Surgeon to the London Hospital; 1, Park crescent, W.

1847 IMAGE, WILLIAM EDMUND, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square. C. 1889.

†1841 JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.

1863 JACKSON, THOMAS VINCENT, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; Whetstone House, Waterloo road south, Wolverhampton.

1883 JACOBSON, WALTER HAMILTON ACLAND, M.A., M.B., M.Ch., Assistant Surgeon and Lecturer on Anatomy to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 66, Great Cumberland place, Hyde Park. *Trans.* 2.

1892 JAMES, EDWIN MATTHEWS, Belgrave Mansions, Grosvenor gardens.

*1883 JENKINS, EDWARD JOHNSTONE, M.D., The Australian Club, Sydney, New South Wales.

†1851 JENNER, SIR WILLIAM, Bart., M.D., G.C.B., D.C.L., LL.D.Cantab., LL.D.Edin., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Greenwood, Bishop's Waltham, Hants. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

Elected

1884 JENNINGS, CHARLES EGERTON, M.S., M.B.

1881 JENNINGS, WILLIAM OSCAR, M.D., 35, Rue Marbœuf, Avenue des Champs-Elysées, Paris.

1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Cancer Hospital, Brompton; 1, Buckingham Palace Mansions.

1883 JESSOP, WALTER H. H., M.B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 73, Harley street.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

†1847 JOHNSON, SIR GEORGE, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; Emeritus Professor of Clinical Medicine; Fellow and Member of the Council of King's College, London; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884-5. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib. Com.* 1860-1. *Trans.* 10. *Pro.* 1.

1881 JOHNSON, GEORGE LINDSAY, M.A., M.D., Cortina, Netherhall gardens, South Hampstead, and 14, Stratford place, Oxford street.

1889 JOHNSON, HAROLD J., Senior Assistant, Gloucester County Asylum.

1889 JOHNSON, RAYMOND, M.B., B.S., Surgeon to Out-Patients at the Great Northern Central Hospital and the Victoria Hospital for Children; 20, Weymouth street. *Trans.* 1.

1884 JOHNSTON, JAMES, M.D., 11, Chester place, Hyde Park square.

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 9, Upper Wimpole street, Cavendish square.

1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.

Elected

*1875 JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, and Fellow of the Senate, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., Wool Exchange.]

1865 JORDAN, FURNEAUX, Consulting Surgeon to the Queen's Hospital, Birmingham; Selly Hill, Birmingham.

1881 JULER, HENRY EDWARD, Ophthalmic Surgeon to St. Mary's Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Ophthalmic Surgeon to the London Lock Hospital; 23, Cavendish square.

1893 KANTHACK, ALFRED A., M.B., 31, Rodney street, Liverpool.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square.

1872 KELLY, CHARLES, M.D., Professor of Hygiene in King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District; Ellesmere, Gratwicke road, Worthing, Sussex.

*1848 KENDELL, DANIEL BURTON, M.B., Thornhill House, Walton, near Wakefield, Yorkshire.

*1890 KERR, J. G. DOUGLAS, M.B., C.M., 6, The Circus, Bath.

1884 KESER, JEAN SAMUEL, M.D., Physician to the French Hospital, Shaftesbury avenue, W.C.; 11, Harley street, Cavendish square.

*1877 KHORY, RUSTOMJEE NASERWANJEE, M.D., Honorary Obstetric Physician to the Bai Motlibari and Sir Dinsha Petil Hospitals, &c.; Hormazd Villa, Khumballa hill, Bombay.

†1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens. C. 1890-91.

1881 KIDD, PERCY, M.A., M.D., Assistant Physician to the Hospital for Consumption, Brompton, and Assistant Physician to the London Hospital; 60, Brook street, Grosvenor square. *Trans.* 4.

Elected

†1851 KINGDON, JOHN ABERNETHY, Consulting Surgeon to the Bank of England; 2, Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1883 KNAPTON, GEORGE, 98, Lewisham High Road, St. John's, S.E.

1888 KYNSEY, WILLIAM RAYMOND, C.M.G., Inspector-General of Hospitals, Colombo, Ceylon.

1889 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch., Assistant Physician to the Swansea Hospital; Winchester House, Swansea, S. Wales.

1891 LANE, HUGH, 11, The Circus, Bath.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Assistant Surgeon to Guy's Hospital and to the Hospital for Sick Children; 8, St. Thomas's street, Southwark. *Trans.* 3.

1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 22, Cavendish square.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. *Referee*, 1885—. *Lib. Com.* 1879-80, 1888—. *Trans.* 2.

*1873 LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Senior Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1890 LAW, EDWARD, M.D., C.M., 35, Harley street, Cavendish square.

1890 LAWRENCE, HENRY CRIPPS, 12, Sussex gardens, Hyde Park.

1888 LAWRENCE, LAURIE ASHER, 125, Harley street, Cavendish square.

Elected

*1890 LAWRIE, EDWARD, M.B., Surgeon Lieutenant-Colonel, Indian Medical Department; Residency Surgeon; Hyderabad, Deccan.

1884 LAWSON, GEORGE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Surgeon to the Royal London Ophthalmic Hospital, and Surgeon to the Middlesex Hospital; 12, Harley street, Cavendish square.

1880 LAYCOCK, GEORGE LOCKWOOD, M.B., C.M., Melbourne, Victoria, Australia.

1892 LAZARUS-BARLOW, WALTER SYDNEY, M.B., The Acacias, Chesterton, Cambridge. *Sci. Com.* 1892—.

1892 LEADAM, WILLIAM WARD, M.D., 80, Gloucester terrace, Hyde Park.

*1886 LEDIARD, HENRY AMBROSE, M.D., Surgeon to the Cumberland Infirmary; 35, Lowther street, Carlisle.

1882 LEDWICH, EDWARD L'ESTRANGE, Anatomist to the Royal College of Surgeons, Ireland; 31, Harcourt street, Dublin.

†1843 LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.

1883 LEESON, JOHN RUDD, M.D., C.M., Clifden House, Twickenham.

1869 LEGG, JOHN WICKHAM, M.D., C. 1886. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.

1886 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., Obstetric Physician to the London Hospital; 60, Wimpole street, Cavendish square. *Trans.* 1.

1872 LIEBREICH, RICHARD (Consulting Ophthalmic Surgeon to St. Thomas's Hospital, London); Paris.

1878 LISTER, SIR JOSEPH, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery at King's College, London; and Senior Surgeon to King's College Hospital; 12, Park crescent, Regent's Park. C. 1892.

Elected

*1872 LITTLE, DAVID, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; Ophthalmic Surgeon to the Manchester Royal Infirmary; Lecturer on Ophthalmology at the Victoria University; 21, St. John street, Manchester.

1891 LITTLE, ERNEST MUIRHEAD, 40, Seymour street, Portman square.

*1889 LITTLE, JAMES, M.D., Physician to the Adelaide Hospital; Consulting Physician to the Rotunda, St. Mark's, Steevens' and the Children's Hospitals; 14, Stephen's Green North, Dublin.

1889 LITTLE, JOHN FLETCHER, M.B., 32, Harley street, Cavendish square.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital; Assistant Surgeon to, and Demonstrator of Operative Surgery at, St. Bartholomew's Hospital; 19, Upper Berkeley street, Portman square. *Trans.* 2.

1860 LONGMORE, SIR THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff (Retired); Foreign Corresponding Member, "Académie de Médecine;" Assoc. Soc. Chir. de Paris; Officer of Legion of Honour; The Paddock, Woolston, Hants. *Trans.* 2.

1881 LUCAS, RICHARD CLEMENT, B.S., M.B., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; Corresponding Member of the "Société de Chirurgie" of Paris; 18, Finsbury square. *Trans.* 2.

1888 LUFF, ARTHUR PEARSON, M.D., B.Sc., Physician to Outpatients and Lecturer on Medical Jurisprudence at St. Mary's Hospital; Official Analyst to the Home Office; 31, Weymouth street, Portland place.

1883 LUND, EDWARD, Emeritus Professor of Surgery, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

Elected

1887 LUSH, PERCY J. F., M.B., 4, Maresfield gardens, Hampstead.

1867 MABERLY, GEORGE FREDERICK, Mailai Valley, Nelson, New Zealand.

1889 MACALISTER, DONALD, M.A., B.Sc., M.D., Physician to Addenbrooke's Hospital; Lecturer on Medicine, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.

†1873 MACCARTHY, JEREMIAH, M.A., Surgeon to the London Hospital and Lecturer on Surgery at the London Hospital Medical College; 15, Finsbury square. C. 1886-7. *Lib. Com.* 1882-5. *Referee*, 1890—.

1867 MAC CORMAC, SIR WILLIAM, M.A., M.Ch., D.Sc., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. C. 1884-5. *Referee*, 1889—. *Trans.* 1.

1887 MACDONALD, GEORGE CHILDS, M.D.

1866 MACGOWAN, ALEXANDER THORBURN, M.D.

1880 MCHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Surgeon to the Royal Eye Hospital, Southwark; 5, Savile row.

*1859 M'INTYRE, JOHN, M.D., LL.D., Odiham, Hants.

1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 79, Wimpole street, Cavendish square.

1881 MACKENZIE, STEPHEN, M.D., Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College; Physician to the Royal London Ophthalmic Hospital; 18, Cavendish square. *Referee*, 1890—. *Trans.* 1.

1876 MACKEY, EDWARD, M.D., Senior Physician to the Royal Alexandra Hospital for Sick Children; Assistant Physician to the Sussex County Hospital; 3, Portland place, Brighton.

Elected

*1854 MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, The Cedars, Gainsborough, Lincolnshire.

1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.

1889 MACLEHOSE, NORMAN MACMILLAN, M.B., C.M., 13, Queen Anne street, Cavendish square.

1893 MCLEOD, KENNETH, M.D., 39, Clanricarde gardens, Bayswater.

1876 MACNAMARA, CHARLES N., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street. C. 1891-2. *Referee*, 1884-90. *Lib. Com.* 1886-90.

1881 MACREADY, JONATHAN FORSTER CHRISTIAN HORACE, Surgeon to the Great Northern Hospital; 51, Queen Anne street, Cavendish square.

1880 MADDICK, EDMUND DISTIN, 2, Chandos street, Cavendish square.

1886 MAGUIRE, ROBERT, M.D., Physician to Out-patients and Joint Lecturer on Pathology at St. Mary's Hospital; Assistant Physician to the Consumption Hospital, Brompton; 4, Seymour street, Portman square. *Sci. Com.* 1889—.

1880 MAKINS, GEORGE HENRY, Assistant Surgeon to St. Thomas's Hospital and Surgeon to the Evelina Hospital for Children; 47, Charles street, Berkeley square. *Trans.* 1.

1885 MALCOLM, JOHN DAVID, M.B., C.M., Surgeon to the Samaritan Free Hospital; 13, Portman street, Portman square. *Trans.* 1.

1891 MANBY, ALAN REEVE, M.D., Surgeon Apothecary to their Royal Highnesses the Prince and Princess of Wales at Sandringham; East Rudham, Norfolk.

Elected

1890 MANSON, PATRICK, M.D., C.M., LL.D., 21, Queen Anne street, Cavendish square.

1888 MAPOTHER, EDWARD DILLON, M.D., 32, Cavendish square.

1855 MARCET, WILLIAM, M.D., F.R.S., Flowermead, Wimbledon Park. C. 1871. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 MARSH, F. HOWARD, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. V.P. 1891-3. *Lib. Com.* 1880-1. *Trans.* 4.

1892 MARTIN, CHRISTOPHER, M.B., C.M., 22, Broad street, Birmingham.

1891 MARTIN, HENRY CHARRINGTON, M.D., 27, Oxford square.

1884 MARTIN, SIDNEY HARRIS COX, M.D., Assistant Physician to University College Hospital, and to the Hospital for Consumption, Brompton; 10, Mansfield street, Portland place.

1892 MASTERS, JOHN ALFRED, M.D., 35, Bruton street, Berkeley square, and 57, Lexham gardens, Kensington.

1883 MAUDSLEY, HENRY CARR, M.D., 22, Collins street, Melbourne, Victoria.

1892 MAUNSELL, HENRY WIDENHAM, M.A., M.D.Dublin, 37, Stanhope gardens, Queen's gate.

1891 MAY, WILLIAM PAGE, M.D., B.Sc., care of the Countess Visconte, Puttenham Priory, Guildford.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1865 MEDWIN, AARON GEORGE, M.D., Consulting Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square.

1891 MERCIER, CHARLES ARTHUR, M.B., Flower House, Catford.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 21, Manchester square. *Trans.* 1.

Elected

1874 MERRIMAN, JOHN J., 45, Kensington square.

1893 MILEY, MILES, M.B., 21, Belsize avenue, Hampstead.

1882 MILLS, JOSEPH, 28, Queen Anne street, Cavendish square.

1887 MIVART, FREDERICK ST. GEORGE, M.D., Beaumont Lodge, Worple road, Wimbledon.

1891 MOLINE, PAUL, M.B., 42, Walton street, Chelsea.

1883 MONEY, ANGEL, M.D.

1873 MOORE, NORMAN, M.D., Physician and Joint Lecturer on Medicine at St. Bartholomew's Hospital; 94, Gloucester place, Portman square. C. 1891-2. *Referee*, 1886-90. *Sci. Com.* 1889—.

1878 MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing Cross Hospital and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. *Trans.* 2.

1891 MORRIS, GRAHAM, Wallington, Surrey.

1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 8, Cavendish square. C. 1888-9. *Referee*, 1882-7. *Trans.* 10.

1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 8, Harley street, Cavendish square. *Sci. Com.* 1889—.

1885 MOTT, FREDERICK WALKER, M.D., Assistant Physician and Lecturer on Physiology, Charing Cross Hospital; 84, Wimpole street, Cavendish square.

†1888 MURRAY, HUBERT MONTAGUE, M.D., 27, Savile row.

1873 MURRAY, J. IVOR, M.D., 24, Huntriss row, Scarborough.

1880 MURRELL, WILLIAM, M.D., Physician to Out-patients, and Lecturer on Materia Medica and Therapeutics at the Westminster Hospital; 17, Welbeck street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.

1892 MYDDELTON-GAVEY, E. HERBERT, 94, Wimpole street, Cavendish square.

Elected

1863 MYERS, ARTHUR BOWEN RICHARDS, late Brigade-Surgeon, Brigade of Guards; 43, Gloucester street, Warwick square. C. 1878-9. *Lib. Com.* 1877.

1882 MYERS, ARTHUR THOMAS, M.D., 2, Manchester square.

1889 NAPIER, FRANCIS HORATIO, M.B.

1881 NALL, SAMUEL, M.B.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine and Psychological Medicine in the University of Melbourne; 21, Spring street, Melbourne, Victoria.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole street, Cavendish square. *Referee*, 1892—.

1868 NICHOLLS, JAMES, M.D., Trenarren, Newquay, Cornwall.

1849 NORMAN, HENRY BURFORD, The Manor House, Drayton, Taunton, Somerset. *Lib. Com.* 1857.

*1847 NOURSE, WILLIAM EDWARD CHARLES, Bouverie House, Exeter.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

1884 OAKES, ARTHUR, M.D.

1880 O'CONNOR, BERNARD, A.B., M.D., Physician to the North London Hospital for Consumption; Greenhill Park, Harlesden.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1880 OGILVIE, GEORGE, B.Sc., M.B., Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 22, Welbeck street, Cavendish square.

1880 OGILVIE, LESLIE, M.B., B.Sc., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.

1891 OGLE, CYRIL, M.A., M.B., 30, Cavendish square.

Elected

1858 OGLE, JOHN WILLIAM, M.A., M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.

*1855 OGLE, WILLIAM, M.A., M.D., late Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.

1860 OGLE, WILLIAM, M.D., Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

*1883 OLIVER, THOMAS, M.A., M.D., Professor of Physiology, University of Durham; and Physician to the Newcastle-on-Tyne Infirmary; 7, Ellison place, Newcastle-on-Tyne. *Trans.* 1.

*1871 O'NEILL, WILLIAM, M.D., late Physician to the Lincoln Lunatic Hospital, 2, Lindum road, Lincoln.

1892 OPENSHAW, T. HORROCKS, M.B., M.S., Assistant Surgeon to, and Senior Demonstrator of Anatomy at, the London Hospital; 16, Wimpole street, Cavendish square.

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. C. 1889–90. *Sci. Com.* 1889—. *Referee*, 1884-8. *Trans.* 6.

1890 ORD, WILLIAM WALLIS, M.D., 2, Queen street, Mayfair.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Physician to the National Hospital for the Paralysed and Epileptic, Queen square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole street. *Trans.* 1.

1885 ORMSBY, L. HEPENSTAL, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.

Elected

1879 OWEN, EDMUND, M.B., Surgeon to, and Joint Lecturer on Surgery at St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street; 64, Great Cumberland place, Hyde park. *Trans.* 3.

1882 OWEN, HERBERT ISAMBARD, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 40, Curzon street, Mayfair. *Bldg. Com.* 1889-92. *Referee* 1893.

1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890-91. *Referee*, 1884-89. *Lib. Com.* 1886-8. *Trans.* 4.

1892 PAGE, HARRY MARMADUKE, 107, London Wall.

1887 PAGET, CHARLES EDWARD, Medical Officer of Health for the County Borough of Salford; North Bentcliffe, Eccles, Lancashire.

†1840 PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; Foreign Associate of the 'Académie de Médecine,' Paris; 5, Park place, Regent's park. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1886 PAGET, STEPHEN, 57, Wimpole street, Cavendish square.

*1858 PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Yore Bank, Ripon, Yorkshire.

1887 PARDINGTON, GEORGE LUCAS, M.D., 47, Mount Pleasant road, Tunbridge Wells.

1873 PARKER, ROBERT WILLIAM, Senior Surgeon to the East London Hospital for Children; Surgeon to the German Hospital; 13, Welbeck street, Cavendish square. C. 1888-9. *Bldg. Com.* 1889-92. *Referee*, 1891—. *Lib. Com.* 1885-87, 1892—. *Ho. Com.* 1892—. *Trans.* 4.

Elected

1885 PARKER, KUSHTON, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

1891 PARKIN, ALFRED, M.S., M.D., 5, Albion street, Hull. *Trans.* 1.

1889 PARSONS, J. INGLIS, M.D., Physician to Out-patients, Chelsea Hospital for Women; 3, Queen street, Mayfair.

1883 PASTEUR, WILLIAM, M.D., Assistant Physician to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 4, Chandos street, Cavendish square.

1891 PATERSON, WILLIAM BROMFIELD, 64, Brook street, Grosvenor square.

1891 PATON, EDWARD PERCY, M.D., 103, Highbury quadrant.

1865 PAVY, FREDERICK WILLIAM, M.D., LL.D., F.R.S., Consulting Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. V.P. 1893—. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Referee*, 1890—. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889—.

1879 PEEL, ROBERT, 120, Collins street east, Melbourne, Victoria.

1856 PEIRCE, RICHARD KING, Laggan House, Maidenhead.

*1855 PEMBERTON, OLIVER, Consulting Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; H.M. Coroner, Birmingham; 65, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, The Cedars, Broadwas-on-Teme, Worcester.

1887 PENROSE, FRANCIS GEORGE, M.D., Assistant Physician to St. George's Hospital; 4, Harley street, Cavendish square. *Sci. Com.* 1889—.

Elected

1890 PERRY, EDWIN COOPER, M.D., Assistant Physician and Demonstrator of Pathology at Guy's Hospital; Superintendent, The College, Guy's Hospital.

*1879 PESIKAKA, HORMASJI DOSABHAI, late Hon. Surgeon to the G. T. Hospital (Bombay); 43, Hornby Road, Bombay.

*1878 PHILIPSON, GEORGE HARE, M.D., M.A., D.C.L., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Royal Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., LL.D., 10, Henrietta street, Cavendish square, W.

1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Palace Court, Notting hill gate.

1888 PHILLIPS, JOHN, M.A., M.D., Assistant Obstetric Physician, King's College Hospital; Physician to the British Lying-in Hospital; 71, Grosvenor street, Grosvenor square. *Trans.* 1.

1889 PHILLIPS, SIDNEY, M.D., Senior Physician to Out-patients and Lecturer on Materia Medica at St. Mary's Hospital, Senior Physician to the London Fever Hospital, and to the Lock Hospital; 62, Upper Berkeley street, Portman square.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. V.P. 1893—. *Referee*, 1882-3, 1891-93. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1879-81.

1891 PIERCE, BEDFORD, M.D., The Retreat, York.

†1841 PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital; Cranbrook, Byculiah park, Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.

1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to, and Pathologist at, Guy's Hospital; 24, St. Thomas's street, Southwark. *Trans.* 1.

Elected

1889 PITTS, BERNARD, M.B., M.C., Assistant Surgeon to St. Thomas's Hospital; 109, Harley street, Cavendish square.

1885 POLAND, JOHN, Surgeon to the Miller Hospital, Greenwich; 4, St. Thomas's street, Southwark.

1884 POLLARD, BILTON, Assistant Surgeon to University College Hospital, Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.

†1845 POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. P. 1886-7. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 5.

1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street, Cavendish square. C. 1890-91. *Referee* 1887-89, 1892—. *Trans.* 2.

1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.

1892 POWELL, HERBERT ANDREWS, M.A., M.D., M.Ch., 9, St. Thomas's street, Winchester.

1867 POWELL, RICHARD DOUGLAS, M.D., Physician Extraordinary to H.M. the Queen; Physician to the Middlesex Hospital; Consulting Physician to the Hospital for Consumption and Diseases of the Chest at Brompton; 62, Wimpole street, Cavendish square. S. (Oct.), 1883-5. C. 1887-8. *Referee* 1879-83, 1886. *Trans.*

Elected

1887 POWER, D'ARCY, M.A., M.B., Demonstrator of Surgery at St. Bartholomew's Hospital; Surgeon to the Victoria Hospital for Children; 26, Bloomsbury square. *Trans.* 2.

1867 POWER, HENRY, Senior Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde Park. C. 1882-3. V.P. 1892-3. *Referee*, 1870-81, 1891—. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1872-8.

1857 †PRIESTLEY, SIR WILLIAM OVEREND, M.D., LL.D., Consulting Physician to King's College Hospital, and to the West London Hospital and the British Lying-in Hospital; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

1883 PRINGLE, JOHN JAMES, M.B., C.M., Assistant Physician to, Lecturer on Practical Medicine, and Physician in Charge of Skin Department at, the Middlesex Hospital; 23, Lower Seymour street, Portman square. *Trans.* 1.

1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 48, Brook street, Grosvenor square. C. 1893—. *Lib. Com.* 1887-93. *Trans.* 1.

†1850 QUAIN, SIR RICHARD, Bart., M.D., (Hon.) M.D.Dublin, LL.D.Ed., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton, and to the Seamen's Hospital, Greenwich; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

1871 RALFE, CHARLES HENRY, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square. C. 1889. *Referee*, 1885-8.

Elected

1857 VON RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1890 RANSOM, WILLIAM BRAMWELL, M.D., Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Consulting Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.

1892 RAYNER, HENRY, M.D., 2, Harley street, Cavendish square.

1891 READ, HENRY GEORGE, 30, Finsbury square, and "Martins," Shipbourne, Kent.

1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.

1891 REECE, RICHARD JAMES, 34, Eardley crescent, South Kensington.

1882 REID, JAMES, M.D., C.B., Resident Physician in Ordinary to H.M. the Queen, Windsor Castle.

1884 REID, THOMAS WHITEHEAD, Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury, Kent.

1891 REMFRY, LEONARD, M.A., M.D., Obstetrical Physician to the Great Northern Central Hospital; 60, Great Cumberland place.

1891 RENDEL, ARTHUR BOWEN, M.A., M.B., B.C., 44, Lancaster gate.

†1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Physician-in-Ordinary to H.M.'s Household; Emeritus Professor of Medicine in University College; Consulting Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.

1881 RICE, GEORGE, M.B., C.M., Sutton, Surrey.

1887 RICHARDSON, GILBERT, M.A., M.D., Hawthorn House, Putney.

1863 RINGER, SYDNEY, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80, 1889—. *Trans.* 6.

Elected

1889 RIVERS, W. H. RIVERS, M.D., Bethlem Hospital, St. George's road, Southwark.

1871 RIVINGTON, WALTER, M.S., Consulting Surgeon to the London Hospital; 95, Wimpole street, Cavendish square. C. 1885-6. *Trans.* 5.

*1871 ROBERTS, DAVID LLOYD, M.D., Obstetric Physician to the Manchester Royal Infirmary; Physician to St. Mary's Hospital, and Lecturer on Clinical Obstetrics and Gynæcology at the Owens College, Manchester; 11, St. John street, Manchester.

1893 ROBERTS, D. WATKIN, M.D., 56, Manchester street, Manchester square.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics, and of Clinical Medicine, in University College, London; and Physician to University College Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square. *Sci. Com.* 1889—.

1889 ROBERTS, H. LESLIE, M.B., C.M., 31, Rodney street, Liverpool.

1889 ROBERTS, SIR WILLIAM, M.D., B.A., F.R.S., 8, Manchester square. *Trans.* 2.

1873 ROBERTSON, WILLIAM HENRY, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital; Buxton, Derbyshire.

*1888 ROBINSON, FREDERICK WILLIAM, M.D., C.M., Huddersfield.

1889 ROBSON, ARTHUR WILLIAM MAYO, Hillary place, Leeds. *Trans.* 2.

1885 ROCKWOOD, WILLIAM GABRIEL, M.D., Colombo, Ceylon.

1890 ROLLESTON, HUMPHRY DAVY, M.A., M.D., Pathologist and Lecturer on Pathology at St. George's Hospital; 13, Upper Wimpole street, Cavendish square.

Elected

1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; and to the Royal Infirmary for Children and Women, Waterloo Bridge road; Oulton Lodge, Aylsham, Norfolk. C. 1879-80.

†1857 ROSE, HENRY COOPER, M.D., Consulting Surgeon to the Hampstead Dispensary; 53, Rosslyn hill, Hampstead. C. 1886-7. *Trans.* 1.

1883 ROSE, WILLIAM, M.B., Professor of Clinical Surgery at King's College; Surgeon to King's College Hospital; and Consulting Surgeon to the Royal Free Hospital; 17, Harley street, Cavendish square.

1889 ROSS, DANIEL MCCLURE, 76, Upper Berkeley street.

1888 ROUGHTON, EDMUND WILKINSON, B.S., M.D., Warden of the College of St. Mary's Hospital; Assistant Surgeon to the Royal Free Hospital; 33, Westbourne terrace, Hyde Park. *Trans.* 1.

1882 ROUTH, AMAND JULES MCCONNEL, M.D., B.S., Physician to the Samaritan Free Hospital for Women; Obstetric Physician to Out-patients, and Lecturer on Practical Midwifery at the Charing Cross Hospital; 14A, Manchester square.

†1849 ROUTH, CHARLES HENRY FELIX, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D., Consulting Surgeon to the Royal Sea-Bathing Infirmary; Union crescent, Margate, Kent.

1882 ROY, CHARLES SMART, M.D., F.R.S., Professor of Pathology in the University of Cambridge; Trinity College, Cambridge.

1891 RUFFER, MARC ARMAND, M.A., M.D., 5, York terrace, Regent's park.

1891 RUSSELL, J. S. RISIEN, M.B., C.M., 4, Queen Anne street, Cavendish square.

Elected

1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1886 SAINSBURY, HARRINGTON, M.D., Physician to the Royal Free Hospital and Assistant Physician to the City of London Hospital for Diseases of the Chest; 63, Welbeck street, Cavendish square. *Trans.* 1.

1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

†1855 SANDERSON, JOHN BURDON, M.D., LL.D., D.C.L. Durham, D.Sc., F.R.S., Waynflete Professor of Physiology in the University of Oxford; 64, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., Surgeon-Major, Medical Officer of Health of the Drayton Union Rural Sanitary District; Surgeon to the Market Drayton Dispensary; and Consulting Physician to the Market Drayton Cottage Hospital; Market Drayton, Shropshire.

1869 SANSOM, ARTHUR ERNEST, M.D., Physician to the London Hospital; Consulting Physician and Vice-President, North-Eastern Hospital for Children; 84, Harley street, Cavendish square. C. 1887-8. *Referee*, 1889—. *Trans.* 2.

1891 DE SANTI, PHILIP ROBERT WILLIAM, 55A, Welbeck street, Cavendish square.

1886 SAUNDBY, ROBERT, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; Professor of Medicine, Mason College; 83A, Edmund street, Birmingham.

†1845 SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; 13A, George street, Hanover square. C. 1872-3.

Elected

1891 SAUNDERS, FREDERICK WILLIAM, M.B., B.C., Chievley House, near Newbury, Berks.

1879 SAVAGE, GEORGE HENRY, M.D., Lecturer on Mental Diseases at Guy's Hospital; 3, Henrietta street, Cavendish square.

1859 SAVORY, SIR WILLIAM, Bart., F.R.S., Surgeon Extraordinary to H.M. the Queen; Consulting Surgeon to St. Bartholomew's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883-4. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 8.

1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street. *Referee*, 1888—. *Sci. Com.* 1889—.

1892 SCHORSTEIN, GUSTAVE, M.A., M.B., B.Ch., D.Ph., 11, Portland place.

1887 SCOTT, HARRY, M.D., 47, St. Ermin's mansions, Westminster.

*1861 SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

1863 SEDGWICK, WILLIAM, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.

1892 DE SEGUNDO, CHARLES SEMPILL, 2, Aldridge road villas, Westbourne park.

1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood road, Cromwell road.

1877 SEMON, FELIX, M.D., Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole street, Cavendish square. *Trans.* 2.

Elected

1882 SHARKEY, SEYMOUR JOHN, M.D., Physician and Joint Lecturer on Pathology at St. Thomas's Hospital; 2, Portland place. *Trans.* 2.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1886 SHAW, LAURISTON ELGIE, M.D., Assistant Physician to Guy's Hospital; 10, St. Thomas's street, Southwark.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.S., Assistant Surgeon, Charing Cross Hospital; 20, Stratford place, Oxford street. *Trans.* 3.

1887 SIDEBOTHAM, EDWARD JOHN, M.B., Erlesdene, Bowdon Cheshire.

†1848 SIEVEKING, SIR EDWARD HENRY, M.D., LL.D., F.S.A., Physician-in-Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's and the Lock Hospitals; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

1886 SILCOCK, ARTHUR QUARRY, M.D., B.S., Surgeon in charge of Out-patients, St. Mary's Hospital; Surgeon, Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square.

†1842 SIMON, SIR JOHN, K.C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

1892 SIMS, FRANCIS MANLEY BOLDERO, 12, Hertford street, Mayfair.

1857 SIORDET, JAMES LEWIS, M.D., Villa Labrolles, Mentone, Alpes Maritimes, France.

1890 SMALE, MORTON, 22A, Cavendish square.

1879 SMITH, E. NOBLE, Surgeon to All Saints' Children's Hospital; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

Elected

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square.

1891 SMITH, G. COCKBURN, M.D., 5, Inverness gardens, Kensington.

†1838 SMITH, HENRY SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

1866 SMITH, HEYWOOD, M.A., M.D., 18, Harley street, Cavendish square.

1886 SMITH, HOWARD LYON, Buckland House, Buckland Newton, Dorchester.

1885 SMITH, JAMES GREIG, M.B., C.M., Lecturer on Surgery, Bristol Medical School; Surgeon to the Bristol Royal Infirmary; 16, Victoria square, Clifton, Bristol.

1889 SMITH, ROBERT PERCY, M.D., B.S., Resident Physician and Medical Superintendent, Bethlem Royal Hospital, St. George's road, Southwark.

1892 SMITH, SOLOMON CHARLES, M.D., 4, Portman Mansions, Baker street.

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.

1872 SMITH, THOMAS GILBART, M.A., M.D., Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. C. 1890. *Trans.* 1.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital Society, Greenwich.

Elected

*1874 SMITH, WILLIAM ROBERT, M.D., D.Sc., Barrister-at-Law, Professor of Forensic Medicine in, and Director of the Laboratories of State Medicine at, King's College, London; Medical Officer to the School Board for London; 74, Great Russell Street. *Trans.* 1.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

1889 SPENCER, HERBERT R., M.D., B.S., Assistant Professor of Midwifery in University College; Assistant Obstetric Physician to University College Hospital; 10, Mansfield street, Portland place.

1887 SPENCER, WALTER GEORGE, M.B., M.S., Assistant Surgeon to the Westminster Hospital; 35, Brook street, Grosvenor square. *Trans.* 2.

1888 SPICER, ROBERT HENRY SCANES, M.D., Physician to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.

1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 6A, Bedford square.

1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.

†1851 SPITTA, ROBERT JOHN, M.D., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 53, Harley street, Cavendish square. *Trans.* 1.

1891 STEVENS, CECIL ROBERT, M.B., B.S., 11, Finborough road, Redcliffe gardens.

1854 STEVENS, HENRY, M.D., late Inspector, Medical Department, Local Government Board, Whitehall; Falcon Lodge, Hampton, Middlesex.

1884 STEWART, EDWARD, M.D., The Farm, Sheffield.

Elected

†1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

*1879 STIRLING, EDWARD CHARLES, M.A., M.D., Senior Surgeon to the Adelaide Hospital; Lecturer on Physiology in the University of Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].

†1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1865 STOKES, SIR WILLIAM, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1884 STONHAM, CHARLES, Assistant Surgeon to the Westminster Hospital, and Curator of Anatomical Museum; 4, Harley street, Cavendish square.

1871 STRONG, HENRY JOHN, M.D., Consulting Surgeon to the Croydon General Hospital; Colonnade House, The Steyne, Worthing.

†1863 STURGES, OCTAVIUS, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. V.P. 1889. *Referee*, 1882-8.

†1871 SUTHERLAND, HENRY, M.D., Physician to Newland's House and Otto House Private Asylums; 6, Richmond terrace, Whitehall.

1883 SUTTON, JOHN BLAND, Assistant Surgeon to the Middlesex Hospital; 48, Queen Anne street, Cavendish square. *Trans.* 6.

1890 SYERS, HENRY WALTER, M.D., 3, Devonshire street, Portland place.

1886 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; 26, Weymouth street, Portland place.

*1890 SYMPSON, E. MANSEL, M.A., M.D., B.C., 3, James street, Lincoln.

Elected

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 6.

1875 TAY, WARREN, Senior Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital; Consulting Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Diseases of the Skin, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., *Hon. Secretary;* Physician to, and Lecturer on Medicine at, Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 20, Wimpole street, Cavendish square. S. 1889-93. *Sci. Com.* 1889—. *Referee,* 1887-8. *Trans.* 2.

1893 TAYLOR, JAMES, M.D., 34, Welbeck street, Cavendish square.

1890 TAYLOR, SEYMOUR, M.D., Assistant Physician West London Hospital; 16, Seymour street, Portman square.

1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.

1859 TEGART, EDWARD, 60, Scarsdale Villas, Kensington. C. 1888-9.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. C. 1893—. *Trans.* 11.

1890 THOMAS, WILLIAM ROBERT, M.D., Little Forest, Bath road, Bournemouth.

1862 THOMPSON, EDMUND SYMES, M.D., Consulting Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889—. *Referee,* 1876-7. *Trans.* 1.

†1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Member of the "Société de Chirurgie," Paris; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 8.

Elected

1862 THOMPSON, REGINALD EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1891 THOMSON, JOHN ROBERTS, M.D., Monkchester, Bournemouth.

1892 THOMSON, ST. CLAIR, M.D., 28, Queen Anne street, W.

1881 THOMSON, WILLIAM SINCLAIR, M.D., Hon. Surgeon, Kensington Dispensary; late Senior Consulting Surgeon to Peterborough Hospital, and Medical Officer of Health for Peterborough; 1, Palace court, Notting Hill gate.

1892 THORNE, WILLIAM BEZLY, M.D., 5, Gledhow gardens, South Kensington.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Consulting Surgeon to the Grosvenor Hospital for Women and the New Hospital for Women; 49, Montagu square. C. 1891. *Lib. Com.* 1886-90, 1893—. *Trans.* 5.

1883 THURSFIELD, THOMAS WILLIAM, M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.

†1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-81.

1889 TIRARD, NESTOR ISIDORE CHARLES, M.D., Professor of Materia Medica and Therapeutics, King's College; Physician to King's College Hospital, and Physician to the Evelina Hospital for Sick Children; 28, Weymouth street, Portland place.

1880 TIVY, WILLIAM JAMES, 8, Lansdowne place, Clifton, Bristol.

1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 37, Cavendish square. C. 1887. *Lib. Com.* 1879.

1882 **Tooth**, Howard Henry, M.D., Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Physician to the Metropolitan Hospital; Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 34, Harley street, Cavendish square.

*1871 **Trend**, Theophilus W., M.D., Physician to Royal South Hants Infirmary; 1, Grosvenor square, Southampton.

1879 **Treves**, Frederick, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 6, Wimpole street, Cavendish square. *Referee*, 1890—. *Sci. Com.* 1889—. *Trans.* 5.

*1881 **Treves**, William Knight, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 **Trotter**, John William, formerly Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1859 **Truman**, Edwin Thomas, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1889 **Turnbull**, George Lindsay, M.B., Grove House, 76, Ladbroke grove.

1875 **Turner**, Francis Charlewood, M.A., M.D., Physician to the London Hospital, and to the North-Eastern Hospital for Children; 15, Finsbury square.

1873 **Turner**, George Brown, M.D., The Lodge, Hemel Hempsted, Herts.

1882 **Turner**, George Robertson, Visiting Surgeon to the Seamen's Hospital, Greenwich; Assistant Surgeon to, and Lecturer on Anatomy and Joint Lecturer on Practical Surgery at, St. George's Hospital; 49, Green street, Park lane.

1891 **Tweed**, Reginald, M.D., 55, Upper Brook street, Grosvenor square.

1892 **Tweedy**, John, 100, Harley Street, Cavendish square.

1881 **Tyson**, William Joseph, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne Gardens, Folkestone.

Elected

1876 VENN, ALBERT JOHN, M.D., Physician for the Diseases of Women, West London Hospital; 122, Harley street, Cavendish square.

1870 VENNING, EDGCOMBE, 30, Cadogan place.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and Senior Physician to the French Hospital and Dispensary, Shaftesbury Avenue; 19A, Hanover square.

1891 VOELCKER, ARTHUR FRANCIS, M.D., B.S., Pathologist and Curator of the Museum at the Middlesex Hospital; 13, Welbeck street, Cavendish square.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1886 WAINEWRIGHT, BENJAMIN, M.B., C.M., Assistant Surgeon, Charing Cross and Royal Westminster Ophthalmic Hospitals; 67, Grosvenor street, Grosvenor square.

1884 WAKLEY, THOMAS, jun., 5, Queen's Gate, South Kensington.

*1868 WALKER, ROBERT, Honorary Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1887 WALLACE, EDWARD JAMES, M.D., Holmbush, Grove road, Southsea.

1883 WALLER, AUGUSTUS, M.D., F.R.S., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road.

1888 WALLIS, FREDERICK CHARLES, M.B., B.C., 26, Welbeck street, Cavendish square.

1867 WALLIS, GEORGE, Consulting Surgeon to Addenbrooke's Hospital; 6, Hills road, Cambridge.

Elected

1873 WALSHAM, WILLIAM JOHNSON, C.M., Senior Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 79, Harley street, Cavendish square. C. 1888-9. *Lib. Com.* 1882-5. *Trans.* 6.

*1883 WALTERS, JAMES HOPKINS, Surgeon to the Royal Berk shire Hospital; 15, Friar street, Reading.

1886 WARD, ALLAN OGIER, M.D., Lansdowne House, High road, Tottenham.

1890 WARD, ARTHUR HENRY, Surgeon to Out-patients, Lock Hospital; 7, Hertford street, Mayfair.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1891 WARING, HOLBURT JACOB, M.B., B.S., B.Sc., 15, Upper Brook street.

1877 WARNER, FRANCIS, M.D., Physician and Lecturer on Materia Medica and Therapeutics to the London Hospital; 5, Prince of Wales terrace, Kensington Palace, *Trans.* 1.

1889 WASHBOURN, JOHN WYCHENFORD, M.D., Assistant Physician to, Physician in Charge of Electrical Department, Joint Lecturer on Physiology, and Demonstrator of Bacteriology at Guy's Hospital; Physician to the London Fever Hospital; Guy's Hospital.

1861 WATERS, A. T. HOUGHTON, M.D., Consulting Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.

†1861 WATSON, WILLIAM SPENCER, M.B., Surgeon to the Throat Department of the Great Northern Central Hospital; Surgeon to the Royal Eye Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.

1879 DE WATTEVILLE, ARMAND, M.A., M.D., B.Sc., 30, Welbeck street, Cavendish square.

Elected

1892 WEAVER, FREDERICK POYNTON, M.D., Cedar Lawn, Hampstead Heath.

1840 WEBB, WILLIAM WOODHAM, M.D., Neuilly-sur-Seine, France.

†1891 WEBER, FREDERIC PARKES, M.D., 10, Grosvenor street.

1857 WEBER, HERMANN, M.D., Consulting Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889—. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.

†1844 WEGG, WILLIAM, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.

1874 WELLS, HARRY, M.D., San Ysidro, Buenos Ayres, S. America.

†1854 WELLS, SIR THOMAS SPENCER, Bart., Hon. M.D. Dublin, Leyden, Bologna, Charkoff; Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Foreign Associate, "Académie de Médecine," Paris; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 14. *Pro.* 1.

†1842 WEST, CHARLES, M.D., Foreign Associate of the Academy of Medicine of Paris; care of Edmund Owen, Hospital for Sick Children, 49, Great Ormond street. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital; Senior Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. *Lib. Com.* 1892—. *Trans.* 4.

1888 WETHERED, FRANK JOSEPH, M.D., Assistant Physician City of London Hospital for Diseases of the Chest, Victoria Park; 34, Queen Anne street, Cavendish square. *Trans.* 1.

Elected

1882 WHARRY, CHARLES JOHN, M.D., 14, Ewell road, Surbiton, Surrey.

1881 WHARRY, ROBERT, M.D., 6, Gordon square.

1878 WHARTON, HENRY THORNTON, M.A., Senior Honorary Surgeon to the Kilburn Dispensary; "Madresfield," Acol road, Priory road, West Hampstead.

1875 WHIPHAM, THOMAS TILLYER, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square. C. 1892-3.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 144, Sloane street.

1881 WHITE, WILLIAM HALE, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee*, 1888—. *Trans.* 4.

1890 WHITE-COOPER, G. O., M.B., 5, Cranley gardens, Brompton.

*1881 WHITEHEAD, WALTER, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, Manchester and Salford Lock Hospital, and Manchester and Salford Skin Hospital; 499, Oxford road, Manchester. *Trans.* 1.

1885 WHITLA, WILLIAM, M.D., Professor of Materia Medica and Therapeutics, Queen's College, Belfast; Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.

1877 WHITMORE, WILLIAM TICKLE, Senior Surgeon to the Westminster General Dispensary, to the St. George's and St. James's Dispensary, and to the Gordon Hospital for Diseases of the Rectum; 7, Arlington street, Piccadilly.

1852 WIBLIN, JOHN, The Hermitage, Clewer, Windsor. *Trans.* 1.

*1870 WILKIN, JOHN F., M.D., M.C., The Warren, Beckenham, Kent.

Elected

*1883 WILKINSON, THOMAS MARSHALL, late Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; 33, Avenue road, Grantham.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street, Grosvenor square. *Referee*, 1872–81. *Sci. Com.* 1.

*1883 WILLANS, WILLIAM BLUNDELL, Much Hadham, Herts.

1890 WILLCOCKS, FREDERICK, M.D., Physician to Out-Patients, and Lecturer on Materia Medica and Therapeutics, at the Charing Cross Hospital; Physician to the Evelina Hospital for Sick Children; 14, Mandeville place, Manchester square.

†1865 WILLETT, ALFRED, *Trustee*, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. V.P. 1890-91. *Referee*, 1882-89, 1892—. *Bldg. Com.* 1889-92. *Ho. Com.* 1892—. *Trans.* 2.

1887 WILLETT, EDGAR, M.B., 25, Welbeck street, Cavendish square.

1888 WILLIAMS, CAMPBELL, 24, Welbeck street, Cavendish square.

*1859 WILLIAMS, CHARLES, Senior Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., *Trustee for Debenture-holders*, Senior Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee*, 1888—. *Lib. Com.* 1880-3. *Sci. Com.* 1889—. *Trans.* 5.

Elected

1881 WILLIAMS, DAWSON, M.D., Assistant Physician to the East London Hospital for Children; 25, Old Burlington street.

1872 WILLIAMS, JOHN, M.D., Physician Accoucheur to H.R.H. the Princess Beatrice; Professor of Midwifery, University College, London; Obstetric Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1891. *Referee*, 1878-90. *Lib. Com.* 1876-82.

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Regulations relative to the publication of the *Proceedings*.

The *Proceedings* are issued after each Meeting.

They are sent, postage free, to every Fellow of the Society who, *in writing*, expresses a wish to receive them.

They may be had by others at the Society's House, on payment (in advance) of an annual subscription of five shillings and eightpence, which will include the expense of conveyance by post to any place within the Postal Union. For places beyond the Postal Union special arrangements must be made.

An Abstract of every paper read will appear in the *Proceedings*. Authors will be at liberty, on sending their communications, to intimate to the Secretaries whether they wish them to appear in the *Proceedings* only, or in the *Proceedings* and *Transactions*; and in all cases they must furnish an Abstract of the communication.

The Abstracts of the papers read are sent to the Journals.

ANNUAL GENERAL MEETING,

Wednesday, March 1st, 1893,

At 5 p.m.

Sir ANDREW CLARK, Bart., M.D., LL.D., F.R.S.,
President, in the Chair.

FREDERICK TAYLOR, M.D.,
RICKMAN J. GODLEE, M.S., } Hon. Secretaries.

The Minutes of the last Annual General Meeting were read and confirmed.

The PRESIDENT nominated, as Scrutineers, Dr. C. Theodore Williams and Mr. Herbert Page, and requested them to superintend the Ballot.

The PRESIDENT then called upon the Senior Hon. Secretary, Dr. Frederick Taylor, to read the

REPORT OF THE COUNCIL.

The Council has to report a year of continued prosperity and solid progress in every department of the Society's work. The quality of the papers and the discussions at the meetings have fully maintained the reputation of the Society; and the two Committees, which have been at work for some time upon (1) "Suspended

Animation in the Drowned," and (2) upon the "Medical Climatology and Balneology of the United Kingdom," have brought together much valuable information upon these subjects. The following statement of the work of the latter Committee has been received from its Honorary Secretary:

> "The Reports on the Climate of Cornwall, Devonshire, and the Channel Islands are already completed; and those on Hampshire, Dorset, Somerset, Sussex, Kent, and Surrey are approaching completion.
>
> "The Reports on the Baths and Waters of Bath, Strathpeffer, Moffat, Woodhall, Droitwich, and Leamington are completed; and those on Harrogate, Llandrindod, and Nantwich are expected shortly.
>
> "It is proposed to present to the Society, about Easter, portions of both the Climatological and Balneological Reports."

The number of new Fellows elected is not so great as in 1891, but is equal to the average of previous years.

The newly formed House Committee, which succeeded to the Building Committee, has managed the estate of the Society in such a way as not only to maintain the income anticipated, but considerably to increase it.

On reference to the Treasurers' statement of receipts and payments, it would appear that the year's working has resulted in a decrease of £723 in the balance. It must be borne in mind, however, that among the payments are included a sum of £672 3*s*. 3*d*. transferred to Building Account, bills carried over from the previous year amounting to £325, as well as £62 for extraordinary charges, making a total of £1059. There is also, on the other side, a sum of £50, rent due in 1892, but only paid in 1893. The Treasurers have much satisfaction in stating that additional rents to the amount of about £130 may be

expected in future years. Considering all circumstances, and seeing that the Building Account is now closed with the exception of a balance of about £60, not yet certified by the Architect, the Treasurers have reason to hope that next year they may be enabled to ask the Council to appropriate a certain sum to the formation of a "Sinking Fund" or "Deposit Account," to be applied to the reduction of the Debenture debt.

Although the private installation of the electric light has so far been entirely satisfactory, it has been deemed advisable, as a precaution against breakdown, to connect the Society's wires with the mains of the Westminster Electric Supply Corporation, whose current can now be switched on at a moment's notice. This has been done without any expense to the Society.

Very early in their year of office the Council had to deplore the death of one of their colleagues, the late Dr. Brace, whose health became impaired so soon after his election as to prevent him from attending a single meeting. Dr. Eastes was elected to fill his place.

Mr. Christopher Heath, one of the Trustees, having tendered his resignation, Mr. Alfred Willett was appointed to succeed him.

The time having again come round for the award of the Marshall Hall Memorial Prize, the following Committee was appointed by the Council to advise it:

Sir Andrew Clark, M.D., LL.D., F.R.S.,
Frederick Taylor, M.D.,
R. J. Godlee, M.S.,
William Selby Church, M.D.,
Norman Moore, M.D.,
Stephen Mackenzie, M.D.,
Marcus Beck, M.S.,
P. H. Pye-Smith, M.D., F.R.S.,
William Watson Cheyne, M.B.,
Samuel Jones Gee, M.D.;

and this Committee has submitted the following report:

Report of the Committee appointed to report to the Council on the Selection of a Marshall Hall Prizeman for 1893.

"Your Committee beg leave to report that after full and careful consideration they have unanimously decided to recommend *Dr. William Richard Gowers, F.R.S.*, to the Council as the recipient of the Marshall Hall Prize to be awarded in 1893.

"Your Committee wish to state that certain difficulties have arisen from the wording of one of the Resolutions by which the Committee is guided ('Proc.,' March 1st, 1872), and beg to suggest a modification of the same.

"The second Resolution at present stands thus:—

'That a Prize be given for the best original work done during the previous five years, and recorded in the English language, in anatomical, physiological, or pathological research relative to the nervous system.'

"Your Committee suggest that it should run as follows:

'That a Prize be given for the best original work published during a period of five years in the English language, in anatomical, physiological, or pathological research relative to the nervous system.

'That the period of five years be that which terminates on the 31st day of December preceding the Annual Meeting at which the award is to be made.'

(Signed) "W. S. Church. Stephen Mackenzie.
Samuel Gee. Marcus Beck.
W. Watson Cheyne. Frederick Taylor.
P. H. Pye-Smith. Rickman J. Godlee."
Norman Moore.

The Council has adopted this report, and now recommends the Society to award the prize, amounting to £80 16*s.* 5*d.*, to Dr. William Richard Gowers.

21 Fellows have died during the past year, and 29 Fellows have been elected.

The roll of the Society now contains 434 Resident and 377 Non-resident, or a total of 811 Fellows.

The Honorary Librarians report as follows:

Report of the Honorary Librarians.

"The use of the Library continues to steadily increase; and although the Council has not yet seen its way to largely increase the annual expenditure on the purchase of books and periodicals, very substantial additions have been made to the Library.

"It is not usual in this report to allude specifically to any donations received, but we feel that this year we are justified in making a departure from the usual custom by referring to the very munificent donation which the Library has received from the President, Sir Andrew Clark, in the shape of a very handsomely bound set of the 'Encyclopædia Britannica.' This is an addition to the Library that has long been desired by many Fellows.

"The nucleus of another valuable addition to the Library has been presented by Dr. Howard Tooth, who gave us the first eleven volumes of the 'Dictionary of National Biography.' The new volumes of this series will be purchased as they appear, and we venture to hope that the gap between vol. xi and vol. xxxiii will be promptly filled up by the generosity of other Fellows.

"As the most recent change in the hours of the Library does not seem to meet the convenience of some of the Fellows, it is proposed, either by circular or some equally direct method, to ascertain the views on this subject of every Fellow using the Library.

"Since our last report we have issued to the Fellows a Supplementary Catalogue, containing the titles of all books received down to the end of 1889, and in addition those of a few later accessions which it was found possible to add. Another supplement, bringing the list of accessions down to date, is in the press, and will be published in a few weeks."

In conclusion, the Council wishes to acknowledge the great assistance it has received in every department of the Society's work from the Resident Librarian, Mr. J. Y. W. MacAlister.

The PRESIDENT then called upon Dr. Hare, the Senior Treasurer, to read the Annual Statement of Accounts (pp. xcvi, xcvii).

After some discussion the PRESIDENT moved, and Mr. BARWELL seconded—"That the Report of the Council together with the Treasurer's audited Statement of Accounts be adopted, and printed in the next volume of *Transactions*." Carried *nem. con.*

The PRESIDENT then delivered the ANNUAL ADDRESS (see p. 1).

Resolved, on the motion of Sir EDWARD SIEVEKING, seconded by Mr. LANGTON—"That the best thanks of the Society be given to the President for his Address, and that he be requested to allow it to be printed in the next volume of the *Transactions*." Carried *nem. con.*

Resolved, on the motion of Mr. WARRINGTON HAWARD, seconded by Dr. POLLOCK—"That the best thanks of the

Society be given to the retiring Vice-Presidents, Dr. George Harley and Mr. Howard Marsh, for their services to the Society during their term of office."

Resolved, on the motion of Mr. TIMOTHY HOLMES, seconded by Dr. STEPHEN MACKENZIE—"That the very hearty thanks of the Society be given to the retiring Honorary Secretary, Dr. Frederick Taylor, for his valuable services to the Society both as Honorary Secretary and as Secretary of the House Committee." Carried *nem. con.*

Dr. TAYLOR replying thanked the Fellows for their cordial vote, and expressed the honour he had felt on being selected to discharge the duties of Secretary. The increase in the work of the Honorary Secretary was not quite in proportion to the increase in the correspondence and stationery alluded to in the criticism on the accounts. The bulk of the extra work fell on the Resident Librarian, as Assistant Secretary, and he cordially concurred in the remarks on this subject, both in the President's Address and by other speakers. It would be impossible to carry on the clerical work of the Society single-handed. He pointed out that the larger share of the work in connection with the building had fallen to Mr. Haward, who occupied the post of senior Secretary for a year longer than he himself had done.

Resolved, on the motion of Mr. GEORGE POLLOCK, seconded by Dr. BOWLES—"That the best thanks of the Society be given to the retiring members of Council, Dr. Galabin, Dr. Norman Moore, Dr. Mitchell Bruce, Dr. Barlow, Sir Joseph Lister, and Mr. Macnamara, for their services to the Society during their term of office."

The PRESIDENT then called upon the Scrutineers for their Report, and they announced the result of the Ballot to be as follows:

President.—Sir Andrew Clark, Bart., M.D., LL.D., F.R.S.

Vice-Presidents.—William Selby Church, M.D.;

Frederick William Pavy, M.D., LL.D., F.R.S.; Thomas Pickering Pick; Henry Power.

Treasurers.—Charles John Hare, M.D.; John Ashton Bostock, C.B.

Honorary Secretaries.—John Mitchell Bruce, M.D.; Rickman J. Godlee, M.S., M.B.

Honorary Librarians.—Samuel Jones Gee, M.D.; John Whitaker Hulke, F.R.S.

Members of Council.—Sidney Coupland, M.D.; Alfred Baynard Duffin, M.D.; Philip Henry Pye-Smith, M.D., F.R.S.; George Thin, M.D.; Thomas Tillyer Whipham, M.B.; William Henry Bennett; J. Neville C. Davies-Colley, M.C.; Alban Henry Griffiths Doran; Alfred Pearce Gould, M.S.; George Eastes, M.B.

A vote of thanks was unanimously passed to the Scrutineers, and this terminated the proceedings.

RECEIPTS.	£ s. d.	£ s. d.
To Balance on 1st January, 1892:		
Cash in hand	172 19 5	
,, at Bankers	1277 10 6	
		1450 9 11
,, *Subscriptions, Fees, &c.:*		
403 Annual Subscriptions at £3 3*s.*	1269 9 0	
37 Entrance Fees at £6 6*s.*	233 2 0	
3 Non-resident Subscriptions at £1 1*s.*	3 3 0	
3 Composition Fees (Life)	68 5 0	
Fines	6 2	
		1574 5 2
,, *Transactions* and *Proceedings:*		
Sold by Messrs. Longmans (*Transactions*)	51 19 1	
,, Librarian (*Proceedings*)	9 9	
,, ,, (Catalogues)	15 0	
		53 3 10
,, *Rents received*		2369 2 9
,, *Interest:*		
On Permanent Endowment Fund	12 14 6	
From Prudential Insurance Co.	33 18 8	
		46 13 2
		£5493 14 10

* This includes £30 for Supplementary Catalogue of Library, 1889–91; printing and posting to the Fellows of the abstracts of all papers about to be

† In this is included the payment of Bills, amounting to £325, for the year

CHARLES J. HARE, J. A. BOSTOCK, } *Treasurers.*

MARSHALL HALL

(TRUSTEES: *Walter Butler Cheadle, M.D.*,

The amount of Stock (Consols) standing to credit of this

CHARLES J. HARE, J. A. BOSTOCK, } *Treasurers.*

PAYMENTS.	£	s.	d.	£	s.	d.
By *Rent, Rates, and Taxes*				223	8	10
„ *Lighting, Cleaning, and Heating*	297	7	3			
Fire Apparatus	14	5	6			
				311	12	9
„ *Repairs, Furniture, &c.*				51	6	2
„ *Meeting Expenses*				55	12	0
„ *Printing, Stationery, and Stamped Envelopes*	*186	6	7			
Stamps (other than the above)	7	0	0			
				193	6	7
„ *Officers and Servants:*						
Salaries and Wages				826	4	8
„ *Library:—Books and Binding*				395	5	2
„ '*Transactions*' and '*Proceedings*'				472	13	2
„ *Extraordinary Charges:*						
Conversazione (Balance)	45	13	6			
Address of Condolence to Royal Family	19	19	0			
Science Committees—On Drowning	4	7	6			
"Spas" Committee	25	6	0			
				95	6	0
„ *Building Account*, transferred to				672	3	3
„ *Debentures*, Interest on				1362	1	6
„ Annuity				34	16	0
„ Accountants' and Auditors' Charges				28	18	9
„ *Bank Charges*, Coupons and General Cheques, &c.				3	1	6
„ *Miscellaneous Payments*				52	4	1
				†4778	0	5
Balance:						
Cash in hand	92	8	7			
At Bankers	623	5	10			
				715	14	5
				£5493	14	10

various Registers, Ledgers, &c., connected with the Debentures; and also the read to the Society.
1891.

Audited and approved:
WOODBURN KIRBY, MUNDY, & Co.,
Chartered Accountants,
19, Birchin Lane, E.C.

MEMORIAL PRIZE FUND.

William Ogle, M.D., and Mr. Thomas Smith.)

Fund on the 9th February, 1893, was ... £672 15 11

Audited and approved:
WOODBURN KIRBY, MUNDY, & Co.,
Chartered Accountants,
19, Birchin Lane, E.C.

RECEIPTS.	£	s.	d.	£	s.	d.
To Proceeds of Issue of Debentures			...	36,000	0	0
„ Proceeds of sale of £3085 16*s.* 1*d.* 2¾% ...			...	3016	7	0
„ Interest			...	42	5	4
„ Amount transferred from the General Account ...	2462	3	3			
Less amount paid D. Nutt 21st July, 1890, on account of General Account	6	17	0	2455	6	3
				41,513	18	7
Special Donations for Permanent Endowment Fund	500	0	0			
Less cost of purchase of Annuity as a condition to a Donation of £300	175	18	0	324	2	0
				£41,838	0	7

CHARLES J. HARE,
J. A. BOSTOCK, } *Treasurers.*

PERMANENT

(TRUSTEES: *Sir Andrew Clark, Bart., M.D., Walter*

New South Wales 4% Inscribed Stock

CHARLES J. HARE,
J. A. BOSTOCK, } *Treasurers.*

PAYMENTS.	£ s. d.	£ s. d.
By Purchase of Premises 20, Hanover Square ...	...	23,000 0 0
„ Electric Installation	...	1475 0 0
„ Building, Decorations, Fixtures, Fittings, &c. ...	14,185 19 11	
Less amount received from Mr. Holmes for President's table presented by him to the Society ...	51 0 0	14,134 19 11
„ Cost of Removal	...	255 0 0
„ Interest on Debentures	...	1827 4 3
„ Interest on Annuity (one year)	...	34 16 0
„ Law charges	...	537 9 2
„ Miscellaneous Payments	...	77 2 9
„ Bank charges and Interest	...	71 5 6
„ Disbursements per J. Y. W. MacAlister in respect of petty current expenses for temporary assistance, postages, telegrams, cabs, &c.	...	101 1 0
		41,513 18 7
Permanent Endowment Fund, Investments for, in New South Wales 4% Inscribed Stock (£282 12*s.* 6*d.*)	...	324 2 0
		£41,838 0 7

Audited and approved:
WOODBURN KIRBY, MUNDY, & Co.,
Chartered Accountants,
19, Birchin Lane, E.C.

21st February, 1893.

ENDOWMENT FUND.

Butler Cheadle, M.D., and Mr. Alfred Willett.)

.. .. £326 7 3

ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its 'Transactions.'

ADDRESS

OF

SIR ANDREW CLARK, Bart.,

M.D., LL.D., F.R.S.,

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 1st, 1893.

To-day brings us to the close of another year of our Society's life and work; and here, between the past and the future, it is natural and proper that we should pause for a little while and consider somewhat of the fulfilment of the year which has passed, and of the promise of the year which is to come. We have settled down into our new house, and although in the settlement we have encountered difficulties, they have been overcome, and the overcoming of them has supplied us with fresh strength and energy. Happily the house has in all ways fulfilled our expectations, and proves both a stately and pleasant place in which to dwell and work.

The running of two accounts—the general and the building accounts—concurrently has created a certain intricacy and some apparent contradictions in the balance-sheet, and has made several of the staunchest friends of the Society alarmed lest its financial condition should be, in reality, less satisfactory than it appears to be. But there are no just grounds for these fears; and it is

probable that the coming year's financial working of the Society will yield a balance sufficient to commence a sinking fund for the paying off of our debenture debt.

It is quite true that we have spent, this year, £50 more than we have received, including, however, items which belong to the previous year; but supposing this deficit to be real, it is not unusual. It is nothing to the excess of expenditure over income which has sometimes occurred, and it does not mean that there is an actual deficit in the Society's assets. The intricacies and apparent discrepancies of our accounts arise in great measure from the method followed in their preparation. Practically they are the mere record of receipts and payments for a year, and anyone conversant with business affairs, and understanding how the years overlap and interlace, must know quite well that no single year's account can adequately or justly represent the financial condition of the Society.

I have gone carefully into the accounts of the Society with its chief officers, and have submitted the results of my investigations to the criticisms of an expert, and I find myself justified in declaring that the Society is in a much better financial condition than it was some few years ago, and that at this time it is not only financially sound but financially prosperous.

First let me show you how we stood a few years ago. In 1882 we were £27 in debt to our bankers, in 1884 we were £176 in debt to our bankers, in 1885 we were £176 in debt to our bankers, and in 1890 our expenditure exceeded our revenue.

Secondly, the accounts now in your hands, and certified by our responsible accountants, will satisfy you that our financial condition is sound.

And thirdly, the following statements will conclusively prove to you that we are prosperous.

Our annual subscriptions, amounting to £1269, are larger this year than in any former year of our Society's existence.

The admission of new members is larger than ever

before. The average number of admissions for the years 1887, 1888, and 1889 is twenty-seven ; the average number of admissions for the years 1890, 1891, and 1892 is forty-five. It is true that the number of admissions into the Society was considerably less last year than in the year preceding it; but this is adequately explained by the fact that the prestige accruing to the Society through the acquisition of its new house attracted a large number of eligible candidates anxious to enjoy the improved circumstances of the Society.

And as a still further illustration of the prosperity of our Society, I may mention that in 1887, 1888, and 1889 the average receipts from entrance fees were £171 ; and that in 1890, 1891, and 1892 the average receipts from entrance fees were £287.

The high character, both in originality and in importance, of the papers read before the Society, and of the discussions to which they led, has been adverted to by the Council in its report. Those papers were for the greater part surgical; and although the paucity of the medical papers was in some degree redeemed by the excellence of such communications as those of Dr. Dickinson and Dr. Thin, it is much to be hoped that the distinguished physicians in the ranks of our Fellows will give us, in the coming year, somewhat more of the results of their work and thought.

The number of Fellows lost to the Society by death is twenty-six, a larger mortality than has been ever before recorded.

And now, according to the custom of the Society, I proceed to read the obituary notices. But the Society must forgive me for saying that it is a custom which I do not regard with favour. The reading occupies the place of subjects of more immediate importance ; the histories related have been substantially published beforehand; and the obituaries themselves, laborious to prepare, are, to most of the Fellows, wearisome to hear. It is true that the Society, naturally and excusably, might

wish to preserve in its own 'Transactions' the records of its deceased Fellows. If so, the obituaries, subject to the approval of the Council, might be printed without being read; and to-day, as the reading of the obituaries in full would doubtless exhaust your patience and interfere with the freedom of discussion usual on such occasions, I shall take the liberty to abridge some or to omit others as may seem best.

Walter Hayle Walshe was born in Dublin in 1812. In 1827 he entered Trinity College as a student, and soon developed a linguistic talent so remarkable that he was advised to devote himself entirely to philology. This he determined to do, and with the object of acquiring Oriental languages he proceeded to Paris. Here, however, he found the turning-point in his career, and abandoned philology for medicine. He commenced his studies in 1832, attending La Charité and La Pitié, and learning from the lips of such masters as Louis and Andral, Cruveilhier and Dupuytren. It is interesting to note that amongst his fellow-students at this time was Oliver Wendell Holmes, and between the two there arose a friendship which was interrupted only by death. When Dr. Walshe had completed his medical studies in Paris, he returned to this country and perfected his clinical training at Edinburgh under the teaching of Allison, Christison, and Syme. In 1836 he obtained the M.D. degree of Edinburgh, and soon after his graduation he began medical practice in the north of London. Dr. Walshe now specially devoted his attention to pathology, and published a series of masterly papers on that subject in the 'Cyclopædia of Surgery.' These papers attracted great attention, and soon brought their writer a reputation wide-spread and well deserved. In 1841 he was elected Professor of Morbid Anatomy at University College, where, a few years later, he became, first, special Professor of Clinical Medicine, and then Professor of the Principles and Practice of Medicine. Dr. Walshe also held the post of Physician to University College Hospital, and in 1862,

when he gave up his hospital appointment and his chair, he was elected Consulting Physician and Emeritus Professor of Medicine and Clinical Medicine. He was also for some years Physician to the Brompton Hospital, and on his retirement he was elected Consulting Physician to that institution. Dr. Walshe became a Fellow of the Royal College of Physicians in 1852, and in 1888 the University of Edinburgh conferred upon him the degree of Doctor of Laws. Dr. Walshe was a member of the Medical Society of Observation of Paris, of the Medical Society of Paris, of the Medical Society of Copenhagen, and of the Royal Medical Society of Athens.

Dr. Walshe was a copious and original writer, with a command of easy graceful English which lends an additional attraction to all his works. His earlier contributions to pathology have already been referred to, but from them I may here select for special mention the interesting articles on cancer, cephalhæmatoma, and endarteritis; while from his many contributions to the medical journals in later years I may select the paper on "Adventitious Products," published in 'Todd's Cyclopædia;' the paper on the "Logical Application of Physiology to Pathology"—to which I shall again refer,—published in the 'Medical Times;' and the "Report on Pulmonary Phthisis," published in the 'British and Foreign Medico-Chirurgical Review.' These are all contributions of considerable importance, but the works by which the name of Walshe will be held in remembrance are his two standard treatises on 'Diseases of the Lungs' and on 'Diseases of the Heart and Great Vessels.' Both these works have passed through four editions, and the first mentioned has been translated into French and Danish. Both of them marked a new era in the methodical investigation and in the physical diagnosis of disease. Dr. Walshe was also the translator of 'Magendie's Lectures on the Blood' and of 'Louis's Researches on Phthisis,' one of the most interesting, instructive, valuable, and lasting of books which ever came to us from France.

Dr. Walshe was a keen and accurate observer of facts, and from these his logical and highly trained intellect enabled him to draw conclusions of the greatest value. He it was who first gave us a precise description of moveable kidney and of cephalhæmatoma. He was also among the first to advance the theory that Bright's disease is primarily a disease of the blood, and not of the kidney; and he was one of the earliest to appreciate the modifying influence of diathetic diseases on the course of acute epidemic diseases. Dr. Walshe had teaching talents of the highest order, and alike in his clinical work and in his systematic courses he attained a success and a popularity such as come only to genuine masters of their subject. In Walshe's clinical work the highly developed power of observation, the orderly method of examining patients, and the strictly inductive reasoning by which each conclusion was reached never failed to impress the student profoundly; while in the systematic work he was as much impressed by the extent and variety of the knowledge displayed as he was charmed by the delivery of the lectures in language which was eloquent and yet precise.

In 1850 Dr. Walshe, together with Sir William Jenner, Dr. Parkes, Sir Edward Sieveking, Dr. Hare, and others, founded the London Medical Society of Observation. The main objects of this Society were to promote, by the accurate record of completed cases, clinical knowledge, and to estimate the value and applications of the knowledge thus acquired after the numerical method. The Society published a valuable little book, 'What to Observe;' and it exercised in London and elsewhere a substantial and happy influence on precision of observation, accuracy of narration, the estimation of results, and the methods of their final presentation. Of this Society, which he inspired and in some measure sustained by his personal work and example, Dr. Walshe was President from its birth in 1850 to its attack of suspended animation in 1860. Indeed, the Society is not yet dead, and it requires only the voice of a new and strong master to recall it to life and work.

Through his logical method of clinical investigation Dr. Walshe was enabled to discover and to teach some new and valuable facts in medicine, and the presence of a contracted pupil in aneurism of the aortic arch, the occurrence of sudden death in aortic reflux, and the constitutional origin of some forms of Bright's disease are sufficient illustrations of them.

Dr. Walshe was sincerely opposed to every form of charlatanism; and to that subtle variety of it which, arrayed in the garb of orthodoxy, flaunts along the highway pretending to be good, he offered the most active and implacable opposition.

The address already alluded to (on "The Logical Application of Physiology to Pathology") is generally regarded as one of Dr. Walshe's best performances,—as the one, in truth, in which he reached the highest exercise of his powers of logical exposition and persuasive eloquence. In this lecture, felicitous in language, logical in method, incisive in criticism, sparkling with epigram, and startling with paradox, he declares that no scientific interpretation of the phenomena of disease can be obtained through any knowledge of the structure and functions of the body in health, and that physiology can serve pathology only by supplying standards of comparison and by suggesting plans and measures, observant or experimental, for the investigation of pathological conditions. Furthermore he says, with an emphasis which sends the saying home, that no hypothesis can ever form an actual part of any science.

Now, if any of my hearers will turn from this address to the recently delivered Lumleian Lectures of Dr. Pye-Smith, he will find himself neither surprised nor scandalised when he hears me say, that with all its extensive knowledge and its perfection of logical form, with all its flow and glow of language, all its persuasive eloquence, and all its brilliancy of setting, I regard this address as the most unsound that ever was penned by a distinguished man writing on his own subject. Unless I am hopelessly

befogged, pathology *is* physiology acting merely under altered conditions; and physiology is the only safe and true way to the right understanding of pathological actions and products. And furthermore, when I consider the bold averment that no hypothesis can ever form an actual part of any science, I become filled with amazement, and fear that I have read, thought, and reasoned in vain. Is not the application of the law of gravitation to the explanation of the physical phenomena of the universe an hypothesis? Is it not the biggest of all hypotheses? Does it not embrace infinity? and is it not confessedly inadequate to the solution of all the physical facts of the universe? Is it not merely the best attainable explanation, and may not even the law of gravitation itself be superseded to-morrow by a larger law which shall not be found anywhere at fault? Did not Newton discover many of the leading laws of optics from the adoption of his corpuscular theory of light? Did not Sadi Carnot deduce the law of thermic action still known by his name from an hypothesis respecting the nature of heat, now known to be erroneous? Does not the chemist represent the proportion of weight in which substances combine as atoms of definite weight, and the resulting compounds as definite groups of such atoms? Now this hypothetical coinage has been one of the most useful factors in the progress of chemistry, and yet the symbols are wholly inadequate representations of the facts, and the facts and the symbols are not one. Why, science is instinct with hypotheses; they surround, penetrate, determine, control, and guide it. Without hypotheses science would neither live, move, nor have its being.

The faculty of minute and accurate observation possessed by Dr. Walshe, and his passion for scientific precision, occasionally led him into the dogmatic expression of statements which could not be sustained. Accuracy is the rarest gift granted to men, and even Dr. Walshe was not free from the prime constitutional defect of our race. When, for example, he declared that a patient free from

heart disease, aneurism, or cancer, expectorating half an ounce of blood from the lungs, was certainly tuberculous, he supplies us with a characteristic illustration of this defect.

The least of all the great gifts granted to Dr. Walshe was the practical or empirical gift. He had little therapeutic insight; partly from his cynical nature, partly from his sceptical constitution of mind, and partly from a defect in the recognition of causal relations which were not close together, he had but a narrow belief in the curative efficacy of drugs, and was neither an accomplished nor a successful prescriber.

Furthermore it must be confessed that Dr. Walshe, with apparently almost every qualification entitling him to do so with success, never took any active share in the public life of his profession, and neither this Society nor any of the leading corporations had often the opportunity of being enlightened by his knowledge, benefited by his counsel, or charmed by his eloquence.

Dr. Walshe was altogether an attractive personality. He was engaging in his appearance, he was polished yet kindly in his manners, and his conversation sparkled with humour, epigram, and anecdote. He was a man of most versatile mind and of most varied attainments, and he never was more happy than when bringing all his culture to bear on some favourite hobby. This it was that led to the publication of 'Dramatic Singing, physiologically considered,' in which his ardent admiration of classical music and his scientific knowledge combined to produce a most interesting and instructive work. Only a few years ago he wrote another equally interesting and suggestive book, entitled 'The Colloquial Faculty for Languages and the Nature of Genius,' the last work that proceeded from his facile pen.

For many years Dr. Walshe suffered from a "painful disease" which compelled him to retire from professional work, and to live in a seclusion which was devoted to study. It is to this unhappy circumstance alone that the highly

cultured and eloquent biographer of Dr. Walshe in the pages of the 'Lancet' has ascribed the remarkable seclusion to which I and many others have adverted.

He died on the 14th December, 1892, in the eighty-first year of his age.

Sir William Bowman was born at Nantwich in 1816, and received his early education at Haslewood School, Birmingham. He began his medical studies at the Birmingham General Hospital as an apprentice to Mr. Hodgson. In 1837 he came to London and entered the Medical Department of King's College, where two years later he was appointed Demonstrator of Anatomy. Subsequently he became curator of the museum and prosector; and still later he held the office of Professor of Physiology and of General and Morbid Anatomy. In 1846 he was appointed an Assistant Surgeon to the Royal London Ophthalmic Hospital, and five years later he became full surgeon to that institution. In 1855 he was elected a Fellow of King's College, and in 1877 he became a member of its Council. On the foundation of the Ophthalmological Society in 1880 Bowman was elected President, and in 1881 he was President of the Ophthalmological Section of the seventh International Medical Congress. In 1884 he was created a baronet. He was also elected to the membership of numerous medical and scientific societies, both in this country and abroad, and honorary degrees were conferred upon him by the Universities of Cambridge, Dublin, and Edinburgh. Furthermore he was a prominent Fellow of the Royal Medical and Chirurgical Society, and did good service as a member of the Council and as Vice-President.

Sir William Bowman was the Father of General Anatomy in England, and the brilliant results of his investigations into the structure of the eye, of the kidney, and of the striped muscles were of themselves sufficient to establish a reputation of the highest order. But Bowman had other and equal claims to distinction, for his practical gifts were as great and as fruitful as his scientific gifts.

As an ophthalmic surgeon he occupied a position which was unique. Unrivalled in his knowledge of the ocular structures, in his experience, and in his operative skill, an accurate observer, and cautious in the framing of conclusions, his services were widely sought after, and his opinions were universally respected. With his patients he was kindly and sympathetic, and when in consultation with his medical brethren he was invariably courteous and considerate. Sir William's writings are not so numerous as might perhaps have been expected, but they are of great and permanent value. He never published anything until he was certain of its accuracy; and the work which, with Dr. Todd, he wrote on the 'Physiological Anatomy and Physiology of Man' had for scope, interest, and scientific value no rival at the time of its publication.

Sir William Bowman was a man of pleasing and striking presence. Courteous in his manner to all, he was genial with his friends. Sagacious and practical in thought, skilful and thorough in work, rich in knowledge, ripe in judgment, conscientious in the discharge of his responsibilities, there was to be found in this assemblage of qualifications and gifts the just secret of his successful career. Himself holding the highest and widest views of life, he had no sympathy with any form of narrowness or of intolerance. He protested strongly against the action of the antivivisectionists, charging them with stopping the gate of knowledge, and neither entering themselves nor permitting others to enter. Self-advancement he never sought, and the honours which came to him were the tributes voluntarily paid to his worthily won success.

In 1887 Sir William Bowman began to retire from practice, and this led the profession to testify to its admiration of his life and work by presenting him with his portrait, which was painted by Mr. Ouless. For several years past Sir William Bowman resided at the home which he made for himself in the country near Dorking, and there about this time last year he was

attacked by pneumonia, which in a few days brought to a close this most useful, honorable, and distinguished life.

Dr. Macfarlane studied medicine at the Universities of Edinburgh and Glasgow; and at the latter he obtained the M.B. and C.M. degree with honours in 1867, and in 1872 the M.D., his thesis for which was specially commended. Immediately after his graduation Dr. Macfarlane settled at Polmont, where he soon built up an extensive practice. In 1874 he removed to Kilmarnock. There he was elected Physician to the Kilmarnock Hospital, and quickly acquired one of the largest and most lucrative country practices in Scotland. In addition to his hospital and private work Dr. Macfarlane filled the appointment of surgeon to the Glasgow and South-Western Railway Company, and he was an examiner in Medicine, Clinical Medicine, Public Health, and Medical Jurisprudence in the University of Glasgow. He was also a Justice of the Peace for the county of Ayr.

Unfortunately the strain and anxiety of these numerous duties proved too much for Dr. Macfarlane's health, and lately it became imperative for him to seek out some less harassing career. He decided to leave Ayrshire, which he did amongst general and public expressions of regret, and coming to London he settled in Manchester Square. Many of his old patients from Scotland still continued to consult him, and he soon acquired a large connection. Dr. Macfarlane's chief object, however, in coming to London was to have an opportunity of indulging in his love for literary and scientific work. He entered into the life of the societies with great zest, and in the Royal Medical and Chirurgical he soon became well known. Within the last few years he published contributions on "The Therapeutic Action of Senna Pods," "On Dreaming," on "Habit in Reference to Sleep and Sleeplessness," and a book on "Insomnia and its Therapeutics," which is one of the most valuable treatises we possess on this subject. At the time of his death he was carrying on some interesting investigations into the action of drugs on the cerebral circulation.

Dr. Macfarlane was a physician of the highest character; he was scrupulously thorough in his professional work, kindly and genial in his manners, and sympathetic yet firm with his patients, who soon learned to trust him as a physician and to value him as a friend. His life was one of hard but well-spent labour, and a life that promised to develop into still higher things. This, however, was not te be. He had long suffered from a chronic kidney trouble; and an attack of acute nephritis, setting in in August last, brought his career to a premature close.

Mr. Taylor was born in London in 1814. He studied medicine at St. Bartholomew's Hospital. In 1837 he became a Member of the Royal College of Surgeons, and commenced general practice in the north of London. Mr. Taylor also acted as lecturer on chemistry, both at St. Thomas's Hospital and at the Middlesex Hospital. He was the compiler of the "Catalogue of Concretions" in the Hunterian Museum, and was elected a Fellow of the Royal College of Surgeons in 1870. He was also a Fellow of this Society, of the Chemical Society, and of the Royal Astronomical Society. He was a man of high character and of extensive knowledge, ready in resource and thorough in all his work. For many years Mr. Taylor suffered from chronic bronchitis, and in the spring of 1892 he died at Montreaux.

Mr. Crosse was a son of the celebrated Norwich surgeon, Mr. John Green Crosse. He studied at St. Bartholomew's Hospital, qualified in 1847, and soon thereafter succeeded to his father's practice. Mr. Crosse became Assistant Surgeon and afterwards full Surgeon to the Norfolk and Norwich Hospital, and when in 1888 he resigned his appointment he was elected Consulting Surgeon and Chairman of the Board of Management. Mr. Crosse was a good surgeon, and a man who was popular with all classes in Norwich and the district around. For many years he suffered from severe attacks of gout, and in the beginning of the present year he was attacked by a slow and distressing form of pneumonia, from which he died.

Dr. Spackman was born at Lutterworth in 1819. He studied medicine at the Middlesex Hospital, and received his qualification to practise in 1840. Two years later he took the degree of M.B. of London, and in 1856 he obtained the M.D. of this University. Soon after his qualification Dr. Spackman settled at Harpenden, Herts, where for some time he was associated with the late Mr. Kingston, and where he acquired a large practice. Dr. Spackman was a good type of the wise and high-minded practitioner. He was justly respected by all the medical men in his neighbourhood, and enjoyed the confidence and regard of a numerous circle of patients and friends. About three years ago Dr. Spackman was compelled to retire from practice on account of his failing health, and he died in September last after a long and painful illness.

Mr. Weiss received his medical education at St. Bartholomew's Hospital. He qualified in 1876, and in 1880 he obtained the Fellowship of the Royal College of Surgeons. In 1883 he was appointed Assistant Surgeon to the West London Hospital, and also some time later Surgeon to the Skin Department of the same institution. When a few years ago a preparatory School of Medicine was tried at the West London Hospital, Mr. Weiss took a very active part in its organisation, and the success that for a time attended the scheme was in large measure due to his efforts.

Mr. Weiss from his boyhood had a passion for outdoor sports, and this eventually proved too strong for the ties that bound him to professional work. About eighteen months ago he gave up his hospital appointment and his house in Hanover Square, and went to reside at Ramsgate, so that he might be free to devote himself to his favourite pastime—yachting. When he gave up his work in London Mr. Weiss seemed to be in the best of health, and it was with a shock of surprise that his friends heard last summer the news of his sudden death while yachting off Gosport.

Dr. Darbishire was born in 1846, and in 1864 he entered

Balliol College, Oxford. In University circles he was widely known by his reputation as an oarsman. He rowed stroke for Oxford in the Inter-University contests of 1868, 1869, and 1870, and also in the Oxford race with Harvard in 1869.

After taking the degree of M.A. at Oxford, Dr. Darbishire commenced the study of medicine at St. Bartholomew's. Subsequent to his qualification he visited the Continental schools. On his return to England he began to practise in Kensington, but he soon left London and returned to Oxford, where he was appointed Physician to the Radcliffe Infirmary. He also held the Lichfield Lectureship in Medicine, an Examinership in the University, and the University coronership.

Dr. Darbishire was a modest, an unassuming, and an accomplished English gentleman; cheerful, hospitable, upright, thorough in all his work, and kindly in all ways. He was beloved by the poor of Oxford, to whom in the out-patients' department of the Radcliffe Infirmary he devoted no small part of his time and skill. Among his private patients also he was highly respected and esteemed.

Dr. Darbishire's health began to break down in 1888. He travelled abroad for some time, but without benefit; and returning to England, he died in December last.

Mr. Alfred Baker was born at Birmingham in 1815. He received his early education at King Edward's School. His medical studies were commenced as a pupil at the old College of Medicine in Birmingham, where he was apprenticed to Mr. Ledsam, Senior Surgeon to the Eye Infirmary. After a distinguished career as a student in his native town Mr. Baker came to London, and completed his medical education at St. Bartholomew's Hospital. He qualified in 1837, and immediately returned to Birmingham, where he was appointed House Surgeon, and soon afterwards Honorary Surgeon to the General Hospital. In 1850 Mr. Baker assisted Dr. Bell Fletcher in starting the Sydenham Medical College, and he held

the office of Lecturer on Surgery in that institution until it became amalgamated with Queen's College. Mr. Baker was a Vice-President of the British Medical Association, and he was also President of the Annual Meeting of the Association at Birmingham in 1872. In 1852 he became a Fellow of the Royal College of Surgeons, and served on its Council for eight years.

In 1881 Mr. Baker resigned his appointment as Surgeon to the Birmingham General Hospital. He had held this post for the long period of thirty-three years, and on his retirement the Hospital Committee presented him with his portrait painted by Mr. Frank Holl, and also elected him Consulting Surgeon. Mr. Baker's lively interest in the welfare of the General Hospital did not, however, cease with his retirement from its staff. He was elected first a member and afterwards Chairman of the Managing Committee, and it was due to his suggestion that the Jaffray Hospital was established in the suburbs for the treatment of the more chronic cases.

Mr. Baker was a dexterous and enterprising surgeon, a man of sound judgment and ripe experience. He was held in high esteem by his fellow-citizens, his patients, and his professional brethren. His life ended with a painful internal disease, which he endured with courage and fortitude to the end.

Frederick George Reed was born in the year 1818. He received his medical education as an articled pupil of the late Mr. Luke at the London Hospital. Soon after his qualification Dr. Reed settled in Hertford, where he acquired a very extensive practice, and was held in high esteem by all classes of the community. Dr. Reed was Physician to the Hertfordshire County Infirmary from 1843 to 1856. He obtained the Fellowship of the Royal College of Surgeons in 1847. The degree of M.D. of St. Andrews was conferred upon him in 1849, and in 1857 he became, by examination, a Member of the Royal College of Physicians. He then, on the advice of his friends Sir Benjamin Brodie and Sir William Fergusson,

removed to Hertford Street, Mayfair, where in a short time he secured a select but still considerable connection.

Dr. Reed was a man of high professional skill, of genial and sympathetic manner, and of upright and independent character. He died, after a short illness, on the 11th of last March, but for the preceding fifteen years his failing health had compelled him to abandon active work.

The late *Dr. Brace* obtained his medical education at King's College and at Edinburgh. He became a Fellow of the Royal College of Surgeons of Edinburgh in 1860. Soon thereafter he commenced practice at Bath, where he was appointed Surgeon to the Royal United Hospital and to the Puerperal Charity. Dr. Brace was a competent practitioner, cheerful and sympathetic in his nature, cultured in tastes, and honourable in all that he did. His services were highly esteemed by a wide circle of patients, to all of whom he was as much the valued friend as the skilful physician. Dr. Brace took a great interest in art, and for many years he was an active and energetic member of the committee of the Burlington Fine Art Club. Dr. Brace died suddenly on May 3rd, 1892, in his sixty-eighth year.

Mr. F. le Gros Clark was born in London in 1811, and at the early age of sixteen he commenced his medical studies as an articled pupil under Mr. Travers, then Senior Surgeon to St. Thomas's Hospital.

As a student Mr. Clark's career was highly successful, and at the close of his curriculum he obtained the Cheselden medal for proficiency in surgery and surgical anatomy. He then visited the schools of Dublin, Paris, Berlin, Göttingen, and Edinburgh. In 1839 Mr. Clark was appointed Assistant Surgeon at St. Thomas's, and Lecturer on Descriptive and Surgical Anatomy. In 1853 he became full surgeon to the hospital, and subsequently he was elected to the Chair of Surgery, which he filled with acceptance for the long period of thirty years. When in 1883 Mr. Clark brought his long and successful career

at St. Thomas's to a close by resigning his acting appointments, he was elected Consulting Surgeon to the hospital.

In 1843 Mr. Clark obtained the Fellowship of the Royal College of Surgeons, and in 1864 he became a member of its Council. In 1867 and in 1868 as Hunterian Professor of Surgery and Pathology he delivered a series of lectures on Surgical Diagnosis. In 1872 he was elected Vice-President of the College of Surgeons, and in 1874 he became President. For many years Mr. Clark was a well-known and popular Fellow of this Society, in which he occupied in succession the offices of Secretary and Vice-President.

Mr. Clark was the author of several important works, amongst which may be mentioned his 'Anatomy and Physiology of the Nervous System,' 'Lectures on the Diagnosis of Shock and Visceral Lesions,' 'Outline of Surgery and Surgical Pathology,' the 'Hunterian Oration' of 1875, 'Collected Papers on Surgery, Pathology, and Allied Subjects,' and the Revised Edition of 'Paley's Natural Theology.' He was also the writer of many excellent contributions to the medical journals, and he was the translator and editor of Dupuytren's works on 'Diseases and Injuries of the Bones' and 'Lesions of the Vascular System,' which were published by the Sydenham Society.

Mr. Clark was a surgeon of wide and accurate knowledge, and possessed of great skill. He was a man of striking presence, with a stately and upright figure, clearly cut features, and a courteous and dignified manner. He was highly cultured, kindly in his nature, and loved and trusted alike by his patients, his pupils, and his friends. He was a man of the simplest and strongest Christian faith, and justified his faith in his life. He took an active interest in the defence of Christianity, and some of his apologetic papers are worthy of the highest praise.

Charles Hawkins was born in 1812. He studied medicine at St. George's Hospital, and in 1836 he became a Member of the Royal College of Surgeons. It was about

this time also that he first began to assist Sir Benjamin Brodie in his practice. The connection between the great surgeon and his assistant soon ripened into a strong and intimate friendship, which was terminated only by Brodie's death. Then Mr. Hawkins set himself to the task of editing the autobiography and writings of his friend. This was to him a labour of love, and the conscientious care that he lavished upon it resulted in a work that may well be described as one of the classics of medical literature. Mr. Hawkins was a surgeon of undoubted ability, but nevertheless when he applied for an appointment as Assistant Surgeon at St. George's he was unsuccessful. Doubtless this was a grave disappointment to him, and especially as he felt that his religious belief as a Catholic had been made the reason for his rejection. He never again applied for a position on the staff, but he did not permit his disappointment to interfere with his love for his *alma mater*, and to her service he still continued to devote the best of his skill and ability. He zealously promoted every interest of the Medical School at St. George's, and to the management of the hospital he gave a great part of his time and attention. For several years he was acting Treasurer to the hospital, and when he resigned that office he was elected a Vice-President. Mr. Hawkins was also an active and popular Fellow of this Society, in which he discharged with success the duties of Secretary, Vice-President, and Treasurer.

By the members of his own faith in London Mr. Hawkins was greatly beloved, and they highly valued his professional services. In 1860, when Cardinal Wiseman was seriously ill at Rome, Mr. Hawkins was summoned to attend him, and on that occasion he received a gold medal from Pius IX in recognition of his services. Mr. Hawkins was a man of great business capability, and resolute and conscientious in carrying out whatever he conceived to be his duty. He never married, but he was happy in the universal respect of his professional brethren and the affection of his numerous friends.

In the beginning of last year he was attacked by bronchitis, which was aggravated by his imprudently persisting in an attempt to carry on the duties for which he had become physically unfit. His friends watched over him with anxious care, and for a time it seemed as if their efforts were to be rewarded by his recovery. This, however, was not to be, and in the month of April he passed from amongst us.

Mr. Crookes studied at St. Bartholomew's Hospital as an apprentice to Sir William Lawrence. He became a Member of the Royal College of Surgeons in 1832, and thereafter he acted for many years as Surgeon to the Farringdon Dispensary, and he was also Surgeon to the North London Eye Infirmary. Mr. Crookes gave up practice at a very early age and retired into Kent, where he led a quiet country life. He died at Folkestone last August, in his eighty-second year.

The death of *Samuel Armstrong Lane*, at the mature age of ninety, has removed from our profession one of its oldest and most honoured members. Mr. Lane studied medicine at the Windmill Street School and at St. George's Hospital. In 1829 he became a Member of the Royal College of Surgeons, and in 1843 a Fellow. Very early in his career Mr. Lane acquired a high reputation as an accomplished anatomist and a skilful surgeon. All circumstances, in fact, seemed to point out for him an honourable career on the staff of his *alma mater*. Nevertheless, when he applied for an appointment as Assistant Surgeon his application was rejected. This, it was said, was chiefly due to the all-powerful influence of Sir Benjamin Brodie, who favoured another candidate. The contest unfortunately raised feelings so bitter and enduring as to make it impossible for Mr. Lane to join at any future time the staff at St. George's Hospital.

Mr. Lane, however, was not the man to be deterred from his purpose by obstacles, however great. Debarred from sharing in the official teaching of St. George's, he founded a rival school in its immediate vicinity. He was

fortunate in securing the co-operation of able and enthusiastic colleagues, and despite the numerous difficulties besetting a venture of this kind, the school soon obtained a considerable reputation. The foundation of St. Mary's Hospital followed, and very largely through the efforts made by Mr. Lane. He himself was elected Senior Surgeon, and many of his former colleagues followed him to the new hospital. Mr. Lane was also a member of the surgical staff of the Lock Hospital. He served for several years on the Council of the Royal College of Surgeons, but he declined the Presidency. In this Society Mr. Lane was at one time a well-known Fellow. He was a member of the Council in 1849, and Vice-President in 1865.

Mr. Lane was a man of untiring energy and indomitable resolution. As a surgeon he was skilful, wide in his views, and opposed to every form of specialism. An old pupil writes of him that he was one of the first to practise ovariotomy, but he declined to commit himself to a special career as an abdominal surgeon, although he knew that by doing so he might win wealth and distinction. The same writer also tells us that Mr. Lane once commenced a series of papers on syphilis. Six of these papers had been published in the 'Lancet,' and were attracting great attention, when their author suddenly refused to carry them further; the reason he gave being that they would bring him what he did not wish—a reputation and a fortune as a specialist.

Above all, however, Mr. Lane was a teacher. He had a thorough knowledge of anatomy and surgery, and a gift of clear and interesting exposition. Able and enthusiastic himself, he also possessed in a rare degree the power of rousing the ability and the enthusiasm of his pupils.

With a strong passion for his work, and with such capabilities for carrying it out, we cannot wonder that he achieved a reputation and a success scarcely, if at all, excelled by any of the medical teachers of his time.

Mr. Lane was a gentleman of the olden school; and in his dress always retained the old-fashioned swallow-tailed

coat and black satin stock. In his manners he was singularly dignified and courteous. While he knew how to be firm when occasion required, he was by nature kind and sympathetic, and despite the stormy scenes through which his early career had led him, he succeeded in the end in conciliating even the bitterest of his antagonists. Mr. Lane had long outlived most of his contemporaries, and his declining years were spent quietly and happily in the retirement of a country life.

Sir Richard Owen was born in Lancaster in 1804. He studied medicine at the University of Edinburgh, and subsequently at St. Bartholomew's Hospital. In 1826 he became a Member of the Royal College of Surgeons, and soon thereafter he commenced practice in Serle Street. About this time he wrote several papers on surgical subjects, one of which, on the practicability of tying the internal iliac artery for aneurism, was read before this Society. The turning-point, however, in Owen's career was brought about by his appointment as assistant to Mr. Clift, the Curator of the Hunterian Museum.

Owen now gave up his professional work and devoted himself entirely to scientific studies. The magnificent collection in the Hunterian Museum was a fitting field for the development of his genius, and he soon began to lay the foundations of his fame as a zoologist and a comparative anatomist. In 1834 Owen was elected a Fellow of the Royal Society, and in the same year he was appointed to the chair of Comparative Anatomy at St. Bartholomew's. In the following year he became Hunterian Lecturer at the Royal College of Surgeons, and in 1836 its Professor of Anatomy and Physiology. Meantime he had been applying himself to the gigantic task of compiling a catalogue of the Hunterian Collection. This monumental work was not brought to a completion until the year 1856, and it necessitated the carrying out of innumerable dissections and other investigations, the results of which were published by Owen in the "Proceedings" of various learned societies.

In 1856 Owen's connection with the College of Surgeons ceased, and he was appointed Superintendent of the Department of Natural History at the British Museum. At that time the Natural History Department was still at Great Russell Street, where there was no possibility of the vast collection being properly displayed and utilised. Owen at once set himself to the task of obtaining a suitable building, and did not rest until there arose the magnificent new museum at South Kensington. Hither the collections were removed, and Owen determined to devote the remainder of his life to the labour of examining, classifying, and arranging them in their new abode.

Of Owen's scientific work it is impossible to speak here except in the briefest and most general terms, and I may simply say that it is remarkable alike for its originality, for its thoroughness, and for its comprehensiveness. The mere list of the names of his published papers occupies twenty-eight columns of the Royal Society's Catalogue, and the papers themselves range over the whole domain of natural history, from the simplest of the Invertebrates to the most complex of the Mammalia, from the primitive organisms of the most remote epochs to the highest organic developments of the present era. Owen's industry as an investigator was immense, but he was much more than a mere accumulator of details. He was a philosophic thinker and reasoner, and the generalisations which he drew from his work are of the highest value. For theory he professed to entertain a considerable contempt, and probably it was this attitude of mind, together with his inability to grasp a supreme generalisation, which led him into the bitter and altogether regrettable controversy with Darwin over the "Origin of Species."

Of the character and value of Owen's scientific work, and of the measure of his contributions to the ideas which will give continuity and development to the biological sciences, it is impossible to speak with any profit here and now.

Owen was more than a mere man of science. He was also

a good citizen; and in many schemes for ameliorating the condition of the poor and for improving the condition of public health he was an active and a wise helper.

Owen was honoured by his Sovereign and by his fellow-workers in science, and numerous distinctions were bestowed on him. He was made a C.B. in 1873, and later he became a K.C.B. He belonged to the Prussian "Ordre pour le Mérite," and to the French "Légion d'Honneur." Orders were also conferred upon him by the King of Italy, the King of the Belgians, and the Emperor of Brazil. He was President of the British Association in 1857. He received honorary degrees from the Universities of Oxford, Cambridge, and Dublin, and most of the learned societies of Europe and America elected him to their membership. His last long and painful illness was borne with exemplary patience and fortitude, and in the end of the year, in his quiet house in Richmond Park where he had dwelt so long, he departed hence.

Professor Hofmann, the distinguished German chemist, had many friends in this country, from the fact that he resided in London from the year 1845 to the year 1862. He came to England to be Superintendent of the Royal College of Chemistry, now the Chemistry Department of the School of Mines. In 1855 he was appointed by Government to be a warden at the Royal Mint. He was President of the London Chemical Society in 1861, and was also elected one of the Honorary Fellows of this Society.

Professor Hofmann returned to Germany to occupy the Chair of Chemistry at Bonn, but he had only held that post for a year when he was appointed Professor of Chemistry at the University of Berlin. He was a member of the Scientific Commission for Medical Affairs, and he was also the founder, and, till the time of his death, the President of the German Chemical Society (Deutsche chemische Gesellschaft). Professor Hofmann was a most eminent scientific worker and investigator. He made important contributions to our knowledge of chemistry,

but his best known and most important work was that in connection with the aniline dyes. His discoveries in this department have contributed in no small measure to raise the dyeing industry to its present condition of prosperity and artistic excellence.

Apart from his scientific attainments Professor Hofmann was a man of a most charming personality. His death, which took place very suddenly and unexpectedly on the 5th of May, will be deeply regretted by many in this country as well as in his fatherland, where he was universally honoured.

John Edward Morgan was the second son of the Rev. Morgan Morgan, vicar of Conway, and brother of Sir G. Osborne Morgan and of the Master of Jesus College, Cambridge. Dr. Morgan obtained his early education at Conway and at Shrewsbury. Subsequently he studied at University College, Oxford, where, in 1852, he took classical honours and the degree of B.A., and afterwards that of M.A. Up to this time it had been his intention to enter the Church, but now he determined to become a member of the medical profession. He entered at St. Mary's Hospital as a student, and when his curriculum there was finished he visited the medical schools of France and Germany. In 1861 he graduated as M.B.Oxford, and in the same year he obtained the Membership of the Royal College of Physicians. Soon after his graduation Dr. Morgan commenced practice in Manchester, and in a very short time he was appointed Physician to Salford Hospital. He was also Lecturer on Pathology at the Manchester Royal School of Medicine, and in 1873 he was elected to the Chair of Medicine at Owens College. In 1865 the M.D. degree of Oxford was conferred upon him, and three years later he was promoted to the Fellowship of the Royal College of Physicians. In 1857 he became a member of the Council of the College of Physicians, and he was also offered a censorship, but this he was compelled to decline on account of his failing health.

Dr. Morgan was an able and accomplished physician, and a man of exceptionally wide culture. As a public speaker he was humorous, brilliant, and effective; as a lecturer he was highly successful and highly popular. With him the first duty was always to his chair and to his school, and he was ever ready to devote his time and his energy to advancing any of the interests of the Victoria University. Dr. Morgan had a large share in founding the Manchester Nurses' Training Institution. He also took a very active part in promoting the public health of Manchester, and for some years he was honorary secretary of the Sanitary Association.

From his undergraduate days onwards Dr. Morgan took a lively interest in athletics, and popularly he was widely known by his book entitled 'University Oars.' In this work he gave the results of a critical inquiry into the after health of the men who took part in the Oxford and Cambridge boat races from 1829 to 1869, and appeared to show conclusively that there was no just foundation for the opinion that these men became unhealthy or were short-lived. Dr. Morgan was the author of 'Town Life amongst the Poorest,' 'Reports on the Health of Manchester,' 'The Danger of Deterioration of Race from the too Rapid Increase of Great Cities;' and also of the more purely medical works, 'Hydatids of the Brain,' 'Idiopathic Lateral Sclerosis,' 'The Treatment of Pleurisy and Empyema,' and, in addition, he published in the journals numerous dissertations on medical subjects.

About eighteen months ago Dr. Morgan somewhat suddenly resigned his chair at Owens College, and then his friends learned to their surprise and regret that he was suffering from thoracic aneurism. From this time onward he was more or less confined to bed at his house near Knutsford, in Cheshire, where in the beginning of last September he died.

Henry John Tylden was born in 1857. He was educated at Uppingham, where he gained school scholarships in 1870 and 1873, and in his final year he became captain of

the school. In 1876 he left Uppingham with one of the school exhibitions and proceeded to Oxford, and obtained an open scholarship at Exeter College. At the University he passed through a successful and distinguished undergraduate career, and gained a first class in moderations and in the final school. After taking his degree he remained for some time as a private tutor at Oxford, but in 1882 he determined to devote himself to medicine. He now entered St. Bartholomew's Hospital as a student, and here also he had a most distinguished career. In 1886 he graduated in medicine at Oxford, and obtained the Brackenbury Scholarship. He then visited Vienna, and on his return to England in 1887 he was appointed House Physician at St. Bartholomew's Hospital, and in the following year he was appointed Casualty Physician. In 1888 he took the Murchison Scholarship in Clinical Medicine, became a Member of the Royal College of Physicians, and on his formal admission was specially complimented by the President on account both of his scholarship and of his practical knowledge. In 1889 he was appointed Assistant Physician to the City of London Hospital for Diseases of the Chest.

Dr. Tylden was an able and enthusiastic student of pathology, and in that department of medicine he had already accomplished some notable work. His paper on the conditions met with in pancreatic diabetes, which was brought before this Society, and his article published in 'Nature' on "The Bearing of Pathology upon the Doctrine of Transmission of Acquired Characters," give us an earnest of the high and scholarly work that might have been expected from him had he been spared. Lately Dr. Tylden had turned his attention to the study of typhoid fever, in the hope of working out some method of producing immunity from the disease. Unfortunately in the course of his investigations Dr. Tylden himself contracted an attack of typhoid, whether from accidental inoculation or from infection in the usual way it was impossible to decide. Be this as it may, the attack terminated fatally,

and removed from the ranks of our profession one of the most accomplished scholars, one of the most indefatigable workers, and one of the most promising pathologists of the present generation.

Dr. Abercrombie was born in 1817 in France, where his father was serving as a surgeon in the British Army of Occupation. He was educated at Trowbridge School and at Caius College, Cambridge, where he obtained a mathematical scholarship, and took his degree as senior optime in 1839. He studied medicine at St. George's Hospital, and took the degree of M.B. of Cambridge in 1845, and of M.D. in 1848. He was also elected a Fellow of this Society, and he became a Fellow of the Royal College of Physicians in 1849. About the same time Dr. Abercrombie commenced practice in Cheltenham, where he soon acquired a large connection, and was elected physician to the Cheltenham Hospital.

Dr. Abercrombie always took a keen interest in athletics, and in his undergraduate days he played cricket for the University of Cambridge against Oxford, and also rowed in the Inter-University boat race of 1839. He was an excellent physician, trusted and beloved by his patients and esteemed by all who knew him. In his disposition Dr. Abercrombie was modest and retiring. He never sought publicity in any form, but was content to live a quietly useful and an honourable life.

In 1879 Dr. Abercrombie gave up his practice and his hospital appointment at Cheltenham, and came to reside in London, where he afterwards lived, and where in August last his life came to a close.

Dr. Davies was born in 1818, and studied medicine at the University of Edinburgh, where in 1843 he took his degree and obtained a gold medal for his thesis. He then proceeded to Bath, where in a short time he acquired one of the leading practices, and was appointed Physician to the Royal United Hospitals. Dr. Davies also acted as President of the Bath and Bristol branch of the British Medical Association. And when in 1849 the Annual

Meeting of that Association was held at Bath, he delivered the address on Medicine. Unfortunately Dr. Davies's useful and promising career was brought to a premature close. His health completely broke down whilst he was yet a comparatively young man, and for the last thirty years he had lived in retirement. He died at the age of seventy-four in February last.

William Wood was born in the year 1816. His father was a surgeon in the 79th Infantry, and his mother a daughter of Sir John Ramsden, Bart. Dr. Wood's student days were passed at University College, where he had a most distinguished career. He subsequently proceeded to Paris, and there his attention was specially directed to the study of clinical work. In 1845 he was appointed principal resident medical officer at Bethlem Royal Hospital. About this time the more humane treatment of lunatics was just beginning to be advocated, and to Dr. Wood belongs the honour of having been one of the first to introduce that treatment in this country. In 1848 he obtained the M.D. degree of St. Andrews. In 1852 he resigned his appointment at Bethlem Hospital, and became the proprietor of a private asylum, first at Kensington House and afterwards at the Priory, Roehampton. In 1861 he was elected Visiting Physician to St. Luke's Hospital. This post he held for thirty years, and on his resignation he was appointed Consulting Physician. In 1864 Dr. Wood was elected a Fellow of the Royal College of Physicians, and subsequently he served on several of its committees, where his knowledge of affairs, the thoroughness of his inquiries, his sound judgment, his fine temper, and his clearness and firmness of mind enabled him to be of much service. Dr. Wood was a well-known and popular Fellow of this Society; he served on the Council, and in 1879 he acted as Vice-President. He was also President of the Medico-Psychological Association of Great Britain and Ireland. Dr. Wood was a man who won the esteem and good-will of all who knew him. His nature was kindly and generous,

and to those who came to him in trouble he was always ready with sound advice and substantial assistance. He was enthusiastically devoted to his work, and threw his whole energy into the task of promoting the comfort and welfare of those who were entrusted to him. Dr. Wood rendered important assistance to the reform of the Lunacy laws, and published his opinions on the subject in a valuable and suggestive pamphlet. He was also the author of a small work, 'The Plea of Insanity,' and of several contributions to the medical journals. About the beginning of last year Dr. Wood had an attack of pleurisy and pneumonia, from which he never completely recovered, and after lingering on through the summer in his country house at Mendip he was again attacked by congestion of the lungs in September, and somewhat suddenly passed away.

M. le Docteur Henri Guéneau de Mussy was born in 1814 at Chalon-sur-Saône. He came of an illustrious race, tracing connections with Montbeliard, with Buffon, and with Voltaire. His father was Court Physician to Charles X, and the same office was occupied by another member of the family during the reign of Louis XIV.

Dr. Guéneau de Mussy studied medicine at Paris, where he passed through an exceptionally brilliant and successful career. Soon after its termination he paid his first visit to this country for the purpose of investigating an outbreak of famine fever. In the course of his work he himself contracted a severe attack of the disease. Fortunately, however, he recovered, and returned to France, where he was rewarded by the Cross of the Legion of Honour, and where the publication of his report removed the last lingering doubts concerning the distinction between typhus and typhoid fevers. In 1846 he accompanied Louis Philippe to England, and here he continued to reside for the long period of twenty-five years, acting as the trusted friend and physician of his exiled monarch.

In the professional circles of London Dr. Guéneau

de Mussy soon became widely known and highly popular. He was an accomplished physician, a man of remarkable literary and artistic culture, and the author of many important contributions to medical knowledge. He was a charming companion, courteous and refined in his manners, and amiable and trustworthy in his nature. To the fallen fortunes of the House of Orleans he was always faithful; and even in the darkest days of their adversity and exile he constantly and zealously devoted himself to their service. By Louis Philippe he was held in the highest affection and esteem, and the rest of the members of the Royal Family of France continued throughout his life to testify to the high value which they set upon his long and faithful services. After his death the royal princes laid with their own hands the tributes of their sorrow on his grave at Père la Chaise.

Before leaving London Dr. de Mussy was entertained at a banquet presided over by Sir Thomas Watson, and attended by almost all the distinguished physicians and surgeons of the day. No one present on that occasion will ever forget the charming guest, the genial company, the speech of Sir James Paget, and the venerated President.

In 1871 Dr. de Mussy finally left this country and took up his permanent residence in Paris, where he was shortly afterwards elected a member of the Academy of Medicine, and where he remained till the time of his death, respected and beloved by all who knew him.

James Anderson died on the 28th of February, 1893. Only three days before he appeared to be in the fulness of health and vigour, and his death, totally unexpected and tragically sudden, has thrown a cloud of grief over many of his contemporaries.

He was born in the year 1853 in the parish of Logie-Buchan, Aberdeenshire. He received his education at the Gordon Hospital School in Aberdeen, and his student days were spent at the university of that city. There he passed through a highly distinguished career in arts, and

when the M.A. degree was conferred upon him he took a gold medal and the Murray Scholarship. In his medical curriculum he was equally successful. His name almost invariably stood first in the records of class examinations, and in 1877 he graduated as M.B. and C.M. "with the highest honours." He was then appointed Demonstrator of Anatomy at the University of Aberdeen, and this post he held for two years. Thereafter he went abroad to Berlin and Vienna, where he devoted himself to the study of clinical work, especially in the departments of laryngology and ophthalmology.

In 1880 Dr. Anderson returned to this country, and soon afterwards he was appointed Demonstrator of Anatomy at the London Hospital, where he has since held the offices of Medical Registrar, Assistant Physician, and Lecturer on Pathology. He was also Senior Assistant Physician to the National Hospital for the Paralysed and Epileptic, and for some years he was Assistant Physician at the City of London Hospital for Diseases of the Chest. Dr. Anderson acted as Examiner in Physiology and Natural History at the University of Aberdeen, and he was also a Member of the Examining Board of the Royal Colleges of Physicians and Surgeons. He was elected a Fellow of the Royal College of Physicians in 1887, and he was likewise a Fellow of this Society and of many of the other medical and scientific societies in London. Dr. Anderson acted as joint editor of the 'Ophthalmic Review,' and he contributed many articles on nervous diseases to the medical journals.

Dr. Anderson was a man of exceptional character, ability, and promise. As a teacher he was popular and successful from his undergraduate days in Aberdeen, when circumstances made teaching a necessity, till later years, when it became one of his chief delights. As a physician his thoroughness, his calm judgment, his special skill in nervous diseases, and his wide knowledge of general medicine were earning for him a well-deserved reputation. He had passed through the seed-time and

the summer, and had reached the harvest which he was never to reap.

Dr. Anderson was widely cultured, and possessed a highly artistic taste. He was pure-minded, upright, self-sacrificing, and independent. A trustworthy and a steadfast friend, he was beloved by all who had the privilege to know him. Recently Dr. Anderson suffered a severe bereavement in the death of his mother. She had been early left a widow, and between her and her son there was a bond of sympathy and love altogether exceptional in its closeness and in its strength. She was ever first in his thoughts; and her approbation was the stimulus that urged him on with the work which begot him success. Since her death he has never been quite the same man. Still he seemed in good health up till the evening of the 26th, when he was suddenly attacked by severe diarrhœa and hæmorrhage, followed by syncope. Next morning he was found by his servants in a state of collapse. Some of his medical friends were hastily sent for; and during the day which followed, everything that their care and love could suggest was done for him. But all was done for him in vain; and his life, full of work, of uprightness, of loving-kindness, and of the highest promise, was thus brought to an untimely end.

Among various schemes for the development of the life and work of the Society which occupied the mind of my devoted predecessor, and its still zealous servant, there are two which cannot be overlooked on this occasion.

The first scheme characteristic of the man was designed to widen and deepen the social intercourse of the Fellows; to do away with those feelings of suspicion and distrust with which from want of truer and larger knowledge we are wont to regard each other, and to knit them into that brotherhood of loving-kindness which our Father Harvey in his wisdom told us should characterise the great profession to which we belong. I share with my predecessor the conviction of the inestimable value of developing the

social side of the life of the Society; and if not by costly and sometimes chilling dinners, yet by such opportunities as occasional evening assemblies offer, I hope we may be enabled in goodly numbers to meet together for the cultivation of mutual confidence and good-will, as well as for our common refreshment and pleasure.

The second scheme which occupied the mind of my predecessor was one to which he adverted on more than one occasion, but which he never propounded in any detail. It was a scheme for the amalgamation of the great Medical Societies of London into a Royal Academy of Medicine and Surgery. The scheme was not original. Many years ago it was projected by some of the leading physicians and surgeons of London. For a lengthened period it was continuously, critically, and sometimes very angrily discussed. But at last there arose a great consensus of opinion that the scheme was impracticable, and so it was abandoned. And, indeed, it needs but small acquaintance with the frailties of human nature to be confident that any scheme, however theoretically perfect, for the amalgamation, union, or fusion of the great medical societies which might now be submitted to them would be forthwith again rejected as impracticable; and this rejection would be both complete and decisive in spite of a general and firm conviction that such a union, if it could be accomplished, would prove of growing advantage not only to the societies, but also to all scientific and practical workers, and to the whole body of the profession.

What is the explanation of this? It lies mainly, as I think, in two difficulties which, if not theoretically, are practically insuperable. The first difficulty is the difficulty of inducing the several societies to sink their respective autonomies in the autonomy of the proposed academy. And supposing this first difficulty to be overcome, there would arise the second difficulty, the difficulty of arranging satisfactorily the financial relations between the old societies and the newly formed institution. For whilst

all men wish to procure advantages, only few are ready to pay for them.

The more this subject has recurred to my thoughts, the more regret I have felt that the scheme for the organisation of a Royal Academy of Medicine—calculated to bring together for their common help all the investigations in the various departments of medicine; to economise the expenses of their technical work; to provide a common centre for the collection, collation, comparison, and criticism of their respective researches; to constitute a body sufficiently representative and sufficiently powerful to defend the rights and promote the just interests of the medical profession; and to create a fountain of honour for the reward of all who distinguish themselves in the science or art of physic—should be finally abandoned. And this regret becomes the more keen when a study of the histories of the medical academies of other countries makes it plain that such an abandonment is unnecessary; for I have myself sufficient faith in the practical wisdom and good feeling of the members of the metropolitan medical societies to believe that all the grave difficulties standing in the way of the organisation of a great academy of medicine and surgery might, through judicious negotiation, be overcome by substituting as the motive power the working idea of federation for the unworkable idea of fusion. By working on the lines of federation each society might continue to preserve its autonomy intact, and no conditions would be imposed upon it beyond those essential to the organisation of the institute and the maintenance of its solidarity. Every society would thus possess and exercise a twofold life,—an individual life and a corporate life; and whilst the one would not interfere with the other, both would co-operate in the building up of a great society which would adequately represent the growing importance, power, and dignity of the medical profession.

There is not the time, and this is not the occasion, to propound in detail any scheme of federation. To-day I content myself with this narrow notice of the subject,

but at no distant date I hope to have the opportunity of suggesting for serious consideration the outlines of a plan for the creation, by federation, of a Royal Academy of Medicine and Surgery.

It now remains for me only to convey to you the expression of my grateful thanks for the high honour you have conferred upon me in electing me to this chair, and for your generous forbearance with my imperfect service.

I also owe grateful thanks to my colleagues, Dr. Taylor and Mr. Godlee, who by their zeal, ability, consideration, and devotion to the interests of the Society, have made easy and pleasant the discharge of my official duties.

Nor can this address be closed without reference to the services of the Resident Librarian, Mr. MacAlister, of whom all my predecessors and all my colleagues have spoken in terms of the highest praise. He has not only to discharge the direct duties of his office, which are numerous and difficult, but he has to manage the twenty-five tenants of the Society, to keep them in peace, and to conduct the correspondence common to both. Mr. MacAlister has brought to the discharge of his multifarious duties notable ability, energy, sagacity, knowledge of affairs, and unflagging zeal, and his services have proved in many different ways of great advantage to the Society.

ON AMPUTATION FOR DIABETIC GANGRENE.

BY

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My reasons for asking the indulgence of the Society to an account of a few rather ordinary cases is, first, that the term *diabetic gangrene* is often, somewhat loosely, made to include a variety of different conditions, our knowledge of which has, however, during the last few years been gradually becoming more definite; and, secondly, that the advance of modern surgery should make us reconsider our position with regard to the treatment of its different varieties.

It is probably true that patients with sugar in the urine are more liable than other people to suffer from gangrene, and that when once started the disease with them is likely to run a more rapid and disastrous course; not that diabetes of itself is supposed to be able to produce gangrene, for the determining causes are usually, if not always, those which might occasion it if no sugar were present in the urine. I believe that this applies not only to simple diabetics but also to gouty subjects, who, at the time, are suffering from glycosuria, and to

diabetics who are the subjects of gout; and, at all events, I venture with all humility to doubt the possibility of always discriminating between these three states, and therefore, from the surgeon's point of view, the necessity (I do not say the advisability) of attempting to do so.

No doubt the reason why the gangrene is more active in diabetics is that their tissues are weaker, and thus less able to resist the growth of micro-organisms and the influence of their products, partly also perhaps because their tissues supply a better pabulum for these low forms of life.

And the determining causes of gangrene are many. But those to which attention is particularly directed are three:

1. Inflammatory conditions.
2. Atheroma of vessels.
3. Peripheral neuritis.

1. The inflammatory conditions are familiar to every one, and include such cases as sloughing of the labia, depending primarily upon itching and consequent scratching of the part; rapidly spreading carbuncles, or the rapid sloughing of ulcers such as I saw some years ago in a gouty old gentleman who had both albumen and sugar in the urine; the front of his leg became quickly involved in a superficial slough starting from a previously existing ulcer, but under the influence of opium and a suitable diet, and the application of antiseptic fomentations, the sloughs separated and he recovered. Under the same head could be included the sloughing in a surgical wound which has not been kept aseptic. This condition is also very well known, but I may illustrate it by a case in which I excised the tongue by Kocher's method in a diabetic patient. He had left the hospital apparently almost well, when extensive sloughing set in in the deeper parts of the wound, and he rapidly died of pyæmia.

2. *Atheroma of vessels.*—It appears to be pretty widely

recognised, though not very distinctly stated in English writings, that patients with diabetes are particularly liable to atheromatous changes in their vessels; and several observers have pointed out—especially in Germany—that in most cases of spontaneous gangrene of the lower extremities in diabetics, extensive arterial degeneration is to be suspected. Frerichs,[1] for example, writing in 1884, says, "Sclerosis of vessels in diabetes, sometimes of the valves, was often observed, especially in cases where gout was present." And Billroth has hinted that possibly the *endarteritis proliferans*, in which there is a great overgrowth of the internal coat leading to blocking of the vessel, but with little tendency of the new growth to degenerative changes, described by Winiwarter,[2] may sometimes be the cause of diabetic gangrene. I shall show the vessels from the amputated leg of one of my patients to illustrate the degree to which arterial degeneration may go on.

Some may possibly be inclined to doubt the truth of this assertion, even while admitting the fact that diabetics very often have rigid arteries; but probably none will deny that glycosuria is a very potent *predisposing* cause of atheroma, as it certainly is of beer drinking; and when a beer-drinking diabetic gets atheroma, it is impossible to discriminate between the two causes which are at work. Israel[3] found arterial sclerosis in thirteen out of twenty diabetics who consulted him, and as he made no observations on the other seven, he presumes that a still larger proportion manifested it. There is a man at University College Hospital now aged thirty-one, with marked diabetes, who has taken copiously of beer from the age of seventeen till he found, twelve months ago, that it began

[1] 'Ueber den Diabetes,' von F. F. Frerichs, Berlin, 1884, s. 77.

[2] "Ueber eine eigenthumliche Endarteritis und Endophlebitis mit Gangrän des Fusses," von Felix v. Winiwarter, 'Archiv für klinische Chirurgie' (Langenbeck), 1879, s. 202.

[3] "Einige Beziehungen des Diabetes Mellitus zum Chirurgie," Berlin, 'Klin. Wochenschrift,' 1882, s. 705.

to disagree with him—five or six pints a day was his allowance;—the arteries of his arms are hard and somewhat tortuous, harder indeed than those of any other patient in the ward, twelve of whom I examined, of ages (given below[1]) varying from 23 to 63.

I examined yesterday a girl of 13, who is attending at University College Hospital for diabetes, from the symptoms of which she has suffered since Christmas, 1891. She passes about 90 oz. of urine, with a sp. gr. 1046 and 1048, and is but little amenable to treatment. In March of this year she suffered from neuritis of the right brachial plexus and the left great sciatic nerve, but the pain and tenderness have quite passed away. She has had no knee-jerks for some time, but has no anæsthesia nor hyperæsthesia. Her radial and brachial arteries are straight but very hard and incompressible, though the pulse is very small and feeble. The posterior tibial vessels are also hard, but the pulse in them is very weak. There is no valvular murmur at the heart.

Anyone looking through published accounts of cases with this idea in his mind cannot fail to be struck with the frequency with which, when the pulse is mentioned, it is added that the vessels are hard and incompressible.

I have placed upon the table specimens and micro-

[1] 1. Male, æt. 62. Interstitial nephritis and chronic bronchitis and emphysema. Arteries a little tortuous, but soft.

2. Male, æt. 29. Aortic regurgitation. Arteries soft and straight.

3. Male, æt. 52. Aphasia, albuminuria. Arteries soft and tortuous.

4. Male, æt. 37. Hæmaturia, enlarged prostate, vomiting. Arteries tortuous and rather hard.

5. Male, æt. 39. Lead colic. Arteries not quite straight, but soft.

6. Male, æt. 47. Cirrhosis of liver. Arteries soft and not tortuous.

7. Male, æt. 30. Gangrene of lung. Arteries straight and soft.

8. Male, æt. 27. Aortic and mitral disease; has had double pneumonia. Arteries straight and soft.

9. Male, æt. 24. Jaundice. Arteries straight and soft.

10. Male, æt. 23. Emphysema and bronchitis. Arteries soft and straight.

11. Male, æt. 37. Phthisis, first stage. Arteries rather hard, but straight.

12. Male, æt. 37. Emphysema and bronchitis, alcoholic. Arteries soft and straight.

scopical preparations of some of the arteries of a man of only 24 years of age—a footman—who died in coma at the Brompton Hospital, which show marked overgrowth and some degeneration of the internal coat. Unfortunately the vessels of the leg were not examined. There is no history of this man's habits with regard to alcohol, but his occupation is a suggestive one.

During the writing of this paper I have only had the opportunity of examining *post mortem* the vessels of two other cases of diabetes. Both were young subjects, and neither showed any naked-eye degenerative changes in the vessels; but most unfortunately both of the preparations have been mislaid before they were examined microscopically.

It is to be hoped that in time more evidence will be forthcoming as to the state of the arteries and veins and peripheral nerves in young people suffering from diabetes, because it is to be noted that spontaneous gangrene does not occur—or very rarely occurs—in them before early middle age, and it is to be presumed that this may depend upon the fact that up to that time the vessels are comparatively speaking healthy. I am conscious that this argument is inconclusive, because it may be urged that neither does carbuncle occur in young people, and, besides, the diabetes of old and middle-aged people seems to stand upon a different footing from the acute disease of children and young people. To this, however, it may fairly be replied that the young person with his acute disease may, if he take sufficient care, live to be a middle-aged person with the chronic disease; and as to carbuncles, we do not know at all how much the arterial circulation or thrombosis may have to do with their causation and progress.

3. The subject of peripheral neuritis is a very large one. Even that portion of it which concerns us is by no means limited. Ziemssen,[1] in 1885, seems to have been the first to start the idea that the neuralgic pains of

[1] 'Münchener med. Wochenschrift,' 1885.

diabetics depend upon peripheral neuritis; and Leyden,[1] in 1888, classified and arranged the various forms in which it may occur. In our own country it has been recognised and described by Buzzard and Althaus, and in France by Charcot and others. A very good *résumé* of the whole subject will be found in a paper by M. B. Auché[2] published in 1890. The conclusion of those who have studied the matter appears to be that diabetics are frequently the subjects of peripheral neuritis arising from no other cause except the glycosuria; that the symptoms following from such affections may consist of motor, sensory, trophic, or vaso-motor derangements; and that while they are subject to considerable variations, they often approximate very closely to those of alcoholic neuritis. The peripheral neuritis of diabetics is apparently much more frequently met with in the lower than in the upper extremities, and the variety, or the combination which chiefly concerns the subject of this paper, is that which causes perforating ulcers, usually originating in corns, just as they do in patients suffering from tabes. It may be set down as a combination of the sensory and trophic forms of the disease, and has even been called *tabetic*. It is noteworthy that the knee-jerks are frequently either temporarily or permanently absent in diabetes, but Argyll-Robertson pupil does not appear to have been ever observed.

I do not wish to suggest that when the gangrene starts from a perforating ulcer, only changes in the nerves are to be looked for. Probably, in most cases, there is arterial degeneration as well; and it is not improbable that, when the arterial degeneration is the most important factor, the nerves may also suffer at the same time. Winiwarter, in his paper on "Endarteritis Proliferans," describes not only arteritis and phlebitis of the posterior tibial vessels, but fibrous thickening of the sheath of the

[1] Leyden, 'Die Entzündung der peripheren Nerven,' 1888.

[2] 'Archives de Médecine expérimentale et d'Anatomie pathologique,' 1890, tome ii, p. 635.

posterior tibial nerve. I would only suggest that, in some cases, those in which the gangrene is painful and spreads rapidly, the cause is most likely to be chiefly arterial degeneration; while in those which are almost painless and very chronic, it probably depends upon changes in the nerves. And the deduction with regard to treatment, which I think may be made, is that, if the former class of cases are to be interfered with at all (and if the gangrene be spreading rapidly, I think it is not only justifiable but indicated), amputation at a considerable distance from the seat of the trouble should be practised. The latter class may more safely be left for the chance of separation by natural means, but if the patient's condition be otherwise good, there is very little risk in removing the necrosed part within a short distance of the completely or imperfectly formed line of demarcation.

This opinion is, of course, quite opposed to that of antiquity. It is opposed, I believe, to that of some surgeons at the present day,—for example, to that expressed in one of the latest English works on operative surgery.[1] But I think that when Mr. Treves says, "Diabetes offers an almost positive bar to any kind of operation;" and again, "Diabetic gangrene of a limb is scarcely within the scope of surgical measures; an amputation in such a condition is almost invariably fatal," he is expressing rather the views of a past generation than those of the present time. König[2] (though he does not advocate early and indiscriminate amputations) strikes, I think, a truer note when he says that the fact that the tissues of a diabetic patient are very prone to serve as a nidus for the growth of micro-organisms should only make us doubly careful, when we operate, to keep our wounds aseptic. In truth, the introduction of the antiseptic principle has rendered operations upon diabetics very much less formidable than they used to be when most of the present text-books on surgery were written. The

[1] 'A Manual of Operative Surgery,' by Frederick Treves, 1891, vol. i, p. 18.

[2] 'Centralblatt für Chirurgie,' No. 13, 1887.

weakened tissues may be, and often are, able to withstand the mere mechanical injury of an operation, although they cannot endure this as well as the irritation produced by putrefaction. At the same time it must not be forgotten that no amount of antiseptics can do away with the danger of coma, or heart failure, following an operation, though their wise employment may immensely diminish the chance of sloughing of the flaps.

I have myself seen a patient die from coma after amputation at the knee for gangrene of the foot, in the hands of a distinguished surgeon whose antiseptics are quite above suspicion.

To illustrate the foregoing observations I will read the notes of two or three cases.

Case 1. *Diabetes; gangrene of foot; amputation above knee; recovery.*—T. T—, the patient, is a large, fat, muscular man, unmarried, 48 years of age, a coffee-house keeper by occupation for the last thirty-one years, who has been much exposed to the heat of the kitchen, and for many years has partaken freely of alcohol in one form or another. Up to thirty years of age he used to take as much as five or six pints or more of beer daily; then he stopped this and took to gin and water, and afterwards whiskey and water and claret. He never was intoxicated, but took it constantly and freely for a prolonged period.

The family history yields only two facts of importance, namely, that an uncle died of gangrene of the toes, and that there appears to be phthisis on the mother's side. He himself gives no history of gout, unless some pain, fourteen years ago, in the right foot may have been due to this cause. He does not appear to have ever suffered from a serious illness, except a bad carbuncle five years previously, and some abscesses of the neck at the age of twenty-eight, until he applied at University College Hospital, in September, 1891, for gangrene of the middle toe of the right foot, which had followed a small festering

sore round the quick of the nail of the little toe, contracted twelve weeks before. There appears to have been considerable inflammation of the foot, which at one time extended halfway up the calf, but had subsided again, and the sore on the little toe had partially if not completely healed. The actual gangrene started only a week before his admission (on the 19th of September, 1890), and was caused by an abrasion on the upper surface of the middle toe, five days before. On admission it was black as far as the base, and the foot was red and hyperæmic almost as far as the malleoli; there was a feeble pulse in both anterior and posterior tibial arteries. The temperature was usually a little below 100°, never above.

He was passing, at this time, ninety ounces of urine a day, with a sp. gr. of 1040, containing a trace of albumen, and 22·1 to 30·8 grains of sugar to the ounce, but there was no undue thirst nor appetite; and although he had for some time been perceptibly losing flesh, none of the ordinary symptoms of diabetes were present. On the 23rd he was put upon a strict antidiabetic diet, and on the 25th he was ordered 1 gr. of opium every sixth hour. Notwithstanding which, though the sugar was reduced to from 16·79 to 23·3 grains per ounce, and the total amount of urine to from thirty-eight to fifty-four ounces in the twenty-four hours, the gangrene continued to extend at the rate of about a quarter of an inch a day, so that be the 25th the purplish blush had involved about three quarters of the foot, and three toes were completely mortified. The heart sounds were normal.

No careful observation upon the state of the peripheral nerves was made at the time, nor was any search made for symptoms of tabes. But since the operation, to be described, it was noted that his knee-jerk was sometimes present, sometimes absent. The ulnar nerves were very easily felt, and were, perhaps, harder than natural; they were not tender, and there was no other tenderness on the course of the large nerves. He did not suffer from neuralgia. The vessels of the upper limbs were hard; the

pulse was weak and unequal in the two radials. The pulse in the left posterior tibial was very weak, that in the dorsal artery of the foot could not be felt; but in any case the amount of subcutaneous fat, and the slight œdema now present, would have made that difficult. It seemed to me that the prospect of a line of demarcation forming was not good, and that the best chance would be given him if the limb were to be amputated above the knee. This was accordingly done on September 28th, in the following way. A circular sweep was made round the limb below the patella, and all the soft parts were raised until the femur was reached just above the condyles. The periosteum was then divided and raised for about an inch, when the bone was sawn through; but, as this did not give quite enough covering, the limb was raised in the air, and the periosteum was separated for nearly another inch, the bone being subsequently removed. When the bleeding had been stopped, this tube of periosteum was in the first place closed, so that before the sutures were applied the face of the stump presented a peculiar appearance, as no bone was apparent at all. A small short drainage-tube was inserted in the middle of the incision, which was removed on the third day. Nothing need be said about the progress of the case. Very great care was taken to conduct the operation antiseptically, and the stump behaved as an aseptic stump should do. The temperature only ran to 99·4° on the day after the operation, and after that did not touch 99°. From the 4th to the 15th, indeed, it was constantly subnormal. The pulse was seldom below 90 or above 100. By October 14th the wound had healed, except a superficial granulating surface half an inch long, which had absolutely closed by the end of the month. It is interesting to note that the patient developed a dusky spot on the heel of the opposite foot, while he was lying in bed, but the epidermis over this never separated, and the spot regained its natural colour.

I append a table showing the analysis of the urine, from which it will be seen that this patient's glycosuria

Date.	Quantity in ounces.	Sp. gr.	Urea.	Albumen.	Sugar.		
					Quantity per ounce.	Total in 24 hours.	
					Grains.	Ounces.	
Sept. 21	33 + ?	1040	...	Trace	30·8	2·12 +	Diet unrestricted.
22	66 + ?	1040	...	„	22·1	3·03 +	
23	90	1038	1·6%	„	25·8	4·89	
24	58	1041	2·5%	⅛	27·23	3·27	Diabetic diet 12 noon on 23rd.
25	48	1039	2·4%	⅛	22·35	2·23	Opium gr. j 6tis horis.
26	38	1038	2·8%	⅛	16·79	1·31	Ate a pound of grapes.
27	80	1035	2·3%	⅛	25·8	4·30	
28	54	1034	2·2%	⅛	21·79	2·43	
29	32 + ?	1020	...	⅛	6·55		
30	45	1036	...	⅛	8		
Oct. 1	58	1032	...	⅛	4		
2	48	1030	...	⅛	3		
3	36						
4	48	1025	...	Trace	1·33		
5	50	1020	...	„	None.		
6	42	1026	...	„	Trace.		
8	68	1028	...	„	„		
9	30	1033	3·25%	„	4·63		
10	24						
11	37	1035	...	Trace	6		
12	64	1024	...	„	2		
13	68	1027	...	„	3·75		
14	60	1022	...	„	2		
15	18?	1027	...	None	3		
16	48	1026	...	„	3		
17	58	1021	...	„	2		
18	49	...	...	„			
19	28	1030	...	„	None.		
20	64	1025	...	„	„		
21	58	1017	...	...	„		
26	48	...	...	None	„		
27	—	1027					

was very amenable to treatment. The improvement, when he was taking an antidiabetic diet and four grains of opium in the twenty-four hours, was very great; for a time, indeed, the sugar absolutely disappeared. A certain amount of albumen (usually only a trace, but sometimes as much as one fifth) was present, but the amount did not bear any constant relation to that of the sugar. It is, however, perhaps possible that the case is one not so much of true diabetes as of glycosuria alternating with albuminuria in a gouty subject. He keeps now in pretty

good health (October, 1892), is much stouter; he is rather careful with his diet, but passes urine of high specific gravity, which contains a large quantity of sugar and some albumen. There is slight œdema of the leg.

In the parts removed at the operation the following points are to be noted:

1. Marked changes due to chronic rheumatic arthritis in the knee-joint, namely, a soft velvety condition of the cartilage, lipping and flange-like projections of the bone, and a rather large loose cartilage.

2. Advanced atheromatous changes in the popliteal artery and the arteries of the leg with much calcareous deposit, some of these vessels being rigid tubes, while the anterior tibial was almost completely obstructed.

3. Microscopically the changes in the internal and middle coats were characteristic of ordinary atheroma, not endarteritis proliferans.

4. No obvious pathological changes in the veins or nerves.

The knee-joint showed marked signs of rheumatoid arthritis, viz. nodular overgrowth sufficient to cause lipping or flange-like projections from the edge of the femoral condyles; a finely granular almost velvety appearance of the cartilage; a loose body in the joint one inch by three quarters of an inch in diameter, and from one eighth of an inch to quarter of an inch thick, one surface being rough and bony in appearance, the other being smooth and apparently cartilaginous, the edges, however, being rolled over so as to form a rim all round it; it weighed thirty-seven grains.

The arteries for the length of eighteen inches were dissected out and are now exhibited. The *popliteal artery* forms a rigid tube from the deposit of calcareous material in the middle coat. To the naked eye, a transverse section shows thickened internal and middle coats; the two can be readily separated. The internal coat, whether viewed from the surface or in section, does not show any patches of atheroma, but appears to be uniformly thickened

and thereby rendered opaque. In the middle coat the calcareous deposit does not occur in the form of rings, but irregularly. Here and there it shows through the inner coat. The external coat shows no obvious changes. The naked-eye changes above described apply also to the arteries of the leg and foot. These are most marked in the anterior tibial, which is nearly twice as large as the posterior tibial. The latter and the peroneal are of about equal size.

The *anterior tibial artery* for a distance of nearly half an inch beyond the bifurcation of the popliteal is so much narrowed by thickening and calcareous deposit in its middle coat that the lumen will barely take an ordinary sized probe. Just below this constriction the artery is a little dilated for an inch and a half, and its coats are thinned. This part of the vessel contains some recent blood-clot. The lower half of the anterior tibial artery is also filled with blood-clot, partly decolourised and firmly adherent to the arterial wall. At the lower end the lumen is completely occluded by very firm clot.

Of the terminal branches of the anterior tibial, the *dorsal artery of the foot* forms a hard rigid tube, and its lumen is filled with partly decolourised clot. The other small arteries are much more healthy.

The *posterior tibial artery* between its origin and the place where the peroneal is given off is completely occluded by thickening and contraction of its middle coat. It may be that there is decolourised clot in the centre of the solid cord which the artery here forms, but this is not visible to the naked eye. Just below the occluded portion this artery is dilated, and contains some recent blood-clot, and below this again and to its lower end it contains decolourised clot which fills up the lumen of the vessel almost throughout its whole length.

The *peroneal artery* from its origin onwards is filled with decolourised clot, and is impervious.

The *veins* were not obviously diseased. The inner coat looked opaque.

Microscopic appearances.—Transverse sections of the

popliteal artery show the following appearances. The inner and middle coats are both much thickened. In some places the inner coat is as thick or even thicker than the middle coat. The external coat is also thicker than normal. Along the concave margin of the section here and there may be seen broadly fusiform epithelial cells with large oval nuclei, and occasionally a layer of smaller spherical cells with round nuclei beneath them. These together constitute the *epithelial layer*. The *subepithelial layer* is enormously thickened. It is composed of fibrous tissue made up of very fine fibrils. The inner part of the layer has the fibrous tissue arranged for the most part in bundles which run in a direction transverse to that of the vessel. Between the bundles are some long spindle-shaped nuclei (but these are relatively few in number), and also some round, more darkly-stained nuclei. The nuclei of this layer are in some instances shrivelled so as to leave a clear space around them. The deeper layers show much less regular arrangement of the tissue. The deepest part of all, next to the elastic lamina, is finely granular or almost homogeneous-looking in many places, not unlike the appearance seen in the later stages of atheroma. In this layer, too, there are a few darkly-stained nuclei. There are no longitudinal fibres to be seen.

The *elastic lamina* in many parts of the section shows no obvious changes. Nowhere is it so wavy as in a normal artery. At those points where the subepithelial layer is thickest the elastic lamina becomes lost, gradually thinning down on each side to a fine edge. In other places it appears as if the lamina were split, since a piece of it can be traced into the subepithelial layer, eventually becoming lost in that layer, while another portion continues on in the normal relation to the middle coat. Between the two portions is tissue like the deeper parts of the subepithelial layer in other places.

The *middle coat* is probably a little thicker than normal throughout, though not markedly so except where deposit of calcareous matter (staining very deeply with

logwood) has taken place. Here, too, is a fusiform thickening of the middle coat. The constituents of this coat have the normal appearance and arrangement except as above indicated.

The *outer coat*, thicker than natural, shows a large number of elastic fibres cut transversely and situated in bundles between the circular fibres of the coat, just outside the middle coat. There are no noteworthy changes in the appearance of this coat.

In none of the coats are the appearances indicative of recent inflammation as evidenced by leucocytic infiltration.

The section of the dorsal artery of the foot shows much the same changes; the calcareous deposit in the middle coat is more extensive.

Sections of the tibial and popliteal nerves, stained first with osmic acid and then with logwood, showed no pathological appearances.

For the foregoing notes of the state of the vessels I am indebted to Mr. G. B. White, Surgical Registrar at University College Hospital.

This case, I think, may be fairly adduced in support of amputation, and *high* amputation, for diabetic gangrene of the foot. The reasons for selecting the knee for the seat of amputation are two: first, because the atheromatous changes here described appear frequently to involve the arteries of the leg, but seldom those of the thigh; secondly, because at the knee it is easy to make a good circular amputation with the minimum of interference within the soft parts.

The next case belongs to a somewhat different category.

Case 2. *Diabetes; gangrene of the little toe; amputation of the toe; recovery.*—R. S—, æt. 61, was admitted under my care into University College Hospital on August 22nd, 1885, on account of gangrene of the right little toe. He had had lumbago and was rheumatic, but had not suffered from rheumatic fever, gout, or syphilis; his mother died of phthisis, but there are no other facts

of importance in his family history. He had been a heavy drinker for twenty years, and a very thirsty man for long—going to bed with a jug of barley water beside him,—and his thirst had increased during the last few years before his admission.

He had had a corn on the under and outer surface of the affected toe for a year, which had suppurated and continued to suppurate till about a fortnight before his admission, when it became gangrenous and very painful —the pain shooting up the limb. A line of demarcation had formed, but there was a good deal of brawny redness on the dorsum of the foot. The superficial arteries appeared to be healthy. The urine had a specific gravity of 1041 and 1045, and contained a considerable quantity of sugar.

I removed the toe on August 27th by an oval incision carried through the dusky tissues. Very careful antiseptic precautions were taken. The wound was left completely open; it healed slowly, but healing was almost complete by the time he left the hospital on October 13th. The temperature did not rise above 99·6° after the operation, but was often over 99° in the evening up to September 17th. On August 27th he was put upon antidiabetic diet, and, though there is no note of it, I have no doubt opium was given, with the result that the sugar very much diminished and ultimately completely disappeared, while the amount of urine passed also diminished, but at the time of his discharge varied still from 70 to 80 ounces in the twenty-four hours.

The notes of this case are very imperfect, the state of the vessels being very slightly recorded. The process was very slow, and, although there was a good deal of pain, I think his trouble was allied to that of a perforating ulcer,—a better example of which, however, is given in the following case.

Case 3. *Perforating ulcer of foot; separation of necrosed part.*—On October 28th, 1891, I saw, with Dr.

Brookhouse, of Brockley, a lady, aged 73, who had completely lost the second toe of the right foot, owing to gangrene which had started in June, from a corn which had been suppurating since January, 1891. When the gangrene once started, its progress was very rapid but almost painless, the patient bearing the various incisions, which Dr. Brookhouse had made, almost without complaint. When I saw her there remained a scar on the dorsum and another on the sole of the foot, and in the latter situation were two openings at the end of a fistulous track two inches long. These I laid into one, at which the patient scarcely shrank; by November 17th the wound had almost healed.

The patient is thin, but has, throughout her life, enjoyed excellent health. She has had rheumatic fever, but not gout, and there is no history of gout in the family. She had facial neuralgia and sciatica about ten years ago, but, with this exception, has been remarkably free from any painful diseases, and certainly no others which could be attributed to peripheral neuritis. The knee-jerks in the early part of this year were almost absent, but there did not appear to be any tactile imperfection, or inability to appreciate heat, either in the upper or lower extremities. There does not seem to be any neurotic tendency in the family.

The urine had been examined previously, but no sugar was found till April, 1891. The actual amount has not been estimated, but it does not seem to have varied, nor does the specific gravity of the urine (which for some months past has varied from 1030 to 1038—on two occasions only having fallen to 1024), notwithstanding a strictly antidiabetic diet, and the administration of Liq. Arsenic. Bromati and Codeia in ½ gr. doses, with Ext. Nucis Vom. gr. ¼ t. d.

I obtained a few more facts about this case at a recent examination (October, 1892). Her diabetic symptoms only dated from eighteen months before the onset of the gangrene, and at the present time the urine has a sp. gr. of 1024,

and contains only a trace of albumen and sugar. The wounds are now soundly healed. For a considerable time she has complained of a numb feeling in both legs and feet, and there appear to be irregularly defined patches of partial anæsthesia and hyperæsthesia in both feet and in the lower thirds of the legs. The pulse in the posterior tibial arteries is not to be felt, but both dorsal arteries of the feet are large and remarkably soft. Her radial arteries are also both remarkably soft and straight for a person of her age. All her life she has been in the habit of taking a small amount of beer with her meals, and very occasionally spirit. Her husband died last year of diabetes; I only mention this because I have seen it stated that diabetes has occurred so frequently in husband and wife as to suggest that it is hardly a fortuitous occurrence.

It would be quite improper to generalise from three cases, and the opportunities are not sufficient for one surgeon, unless his clinique be exceptionally large, or the chapter of accidents throws an unusual number of cases in his way, to gain experience enough to form a valuable opinion from what he has seen. The experience of others, moreover, does not really form a very sound basis for coming to a conclusion, chiefly because the phrase "*strictest antiseptic precautions*" has such a very different meaning in different places. But the records of men like Kuster are well worthy of consideration. Kuster is so convinced of the similarity between senile and diabetic gangrene that he describes them all together. He began by amputating below the knee, but became convinced that the higher operation was much more likely to be successful. It is very important to note that out of eleven diabetics five died, four of coma, and one of heart failure; and when it is remembered that coma may follow a slight accident or such a trivial operation as the removal of a cataract, it behoves us to think twice before recommending any operation in a diabetic that is not essential for the saving of life.

In conclusion, I will state again the two points which it is

the object of this paper, if not to prove, at all events to suggest, that a large proportion of the cases of gangrene of the lower extremities of diabetics will be found to depend either on arterial degeneration or on peripheral neuritis; and that in the former class it is probable that the changes in the arteries will extend at least as high as the knee. Whilst, therefore, it is right to amputate in those of the former class if the disease be progressing rapidly, not lower than the knee, those of the latter may either be left alone, or if amputation be undertaken, it need not be at a great distance from the seat of the disease.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 4.)

ON THE ADVANTAGES

OF AN

AMPUTATION THROUGH THE THIGH,

EITHER AS A PRELIMINARY OPERATION TO, OR, IN SOME CASES, *INSTEAD OF*, AMPUTATION THROUGH THE HIP, WHERE THE HIP-JOINT IS ITSELF DISEASED AND THE PATIENT IN VERY BAD CONDITION.[1]

BY

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To illustrate what I have to say, it will be best to take one of the cases in which this mode of treatment was carried out. But it should perhaps be premised that the mode of treatment suggested is only to be used where all other ordinary methods have failed. Thus it is not meant to take the place of the ordinary treatment of hip-joint disease by prolonged rest, by erasion, or excision. It is, however, meant as an alternative mode of treatment to amputation at the hip-joint, where excision has been tried and has failed, or where the patient's serious

[1] For much kind assistance in looking up these cases I am indebted to Mr. Lawford Knaggs, of Leeds, and to one of my former dressers, Mr. John Fawcett.

condition manifestly does not permit recourse to these methods.

In July, 1879, I was asked to see in consultation with Dr. Bentham, of Hackney, a lad, aged fifteen, suffering from acute tubercular osteitis of the left leg and ankle-joint. There was also some vague swelling over the left hip-joint, the exact nature of which it was difficult to be quite certain of, on account of the extremely bad condition of the limb, and of the patient generally. Movements, however, of the limb caused *pain in the hip-joint*, —pain quite independent of that caused by the disease below the knee. The importance of this appears in the subsequent history of the case. The lad's general condition was very serious. He was extremely emaciated; there were many sinuses over the leg, which were discharging profusely; exposed bone could be felt in all the sinuses; the temperature was high, and the pulse rapid and weak. There was a bedsore over the sacrum, and a tendency to bedsore showed itself over other prominent bones. This condition had commenced three months previously, and the lad had steadily been going from bad to worse. I advised his removal to the hospital for amputation. This was done on July 25th, and the following day the leg was removed by a Stokes-Gritti amputation through the knee,—although hip-joint trouble was then existing. I did this, believing that the lad could not stand the shock of amputation at the hip-joint, and that it was possible, when the mass of diseased tissue below the knee was removed, that the hip-joint disease itself might quiet down. It would be placed, at any rate, in a very much more favorable position for cure by the natural processes, than was possible before the amputation.

It was not only the removal of the diseased tissue, however, which prompted this operation. From observation on other cases I had been led to the belief that even where hip-joint disease was the only existing lesion, amputation through the hip was not imperatively called for, and that much good might be done by amputation

through the thigh *below* the disease, either at the knee-joint or just above.

In cases where only morbus coxæ existed this good was obtained—(1) by shortening the limb, and thus reducing the length of the lever, of which the fulcrum was the hip-joint, thus contributing to rest the joint; (2) by removal of a mass of tissue, which acts mainly as blood-*consuming* tissue, while it acts very little as blood-*producing* tissue.

1. The advantages mentioned in the first heading are very great. In an ordinary case of morbus coxæ, the efforts of nature and of the surgeon are both alike directed to fixing the hip-joint, so as to render it as immoveable as possible, and thus to give it rest. Nature herself does this by spasm or rigidity of the muscles controlling the joint. This spasm is a well-known and well-marked sign of early hip-joint disease, causing the pelvis to move with the femur when attempts are made to flex the joint. It must be apparent that very much greater muscular rigidity or spasm will be required to fix the joint, when the lever which moves it is a long one, than when it is short. Hence, nature has to bring about a much more severe and lasting spasm of the muscles controlling the hip in the one case, than the other. This very spasm is in itself probably an exhausting factor in the disease. The effects of it are shown in the "starting pains" at night, produced by partial relaxation of the muscular fibre during sleep, and then sudden and irregular contraction on any slight movement. (The nerve irritation and muscular exhaustion produced by it are probably analogous to the frontal headache, and succeeding dimness of vision, produced by ciliary spasm in a case of hypermetropia.) The surgeon tries to give the limb rest by putting it into splints and extension apparatus of divers forms and arrangement; but it must be apparent to anybody, who has watched a patient on any of these instruments for immobilising the hip, that they succeed in carrying out their intended object only

imperfectly. From this I do not except the plaster, leather, or poroplastic cases which surround the thigh and the body of the patient. A certain amount of play is soon permitted by all these instruments, and then every movement of the pelvis from side to side, however slight it may be, necessarily involves some motion in the hip-joint. This may easily be seen even in the best of such appliances,—I mean the Bryant double hip-splint. Hence in any critical case of hip-joint disease, any plan which makes perfect immobilisation more easy will make the chances of repair in the hip much greater.

It must be apparent that the muscles controlling the hip-joint will have much less work to do if the *weight of the limb*, and the *length of the lever which they move*, are lessened. Hence in hip-joint disease, if the limb is amputated at or just above the knee, the muscles of the hip can fix the joint with far less expenditure of muscular energy than if the limb is left intact. So great is this gain, that I have generally found in practice that no appliance to fix the hip is necessary after such amputation. The stump moves as if it were one bone with the os innominatum in every movement of the pelvis, the spasm of the hip muscles being quite sufficient to immobilise it. I have come to regard this as the most perfect form of immobilisation of the hip-joint possible.

Of course, it is not in every case that even perfect immobilisation suffices to arrest advanced hip-joint disease. So much damage may already have been inflicted that recovery is impossible. In such cases *excision* may be practised with success, after the amputation through the thigh (as in the case which forms the text for these remarks), if the patient possess sufficient power of recovery; or it may be necessary to proceed to the larger operation of *amputation at the hip-joint*, as in sundry of the other cases narrated in this paper. If *excision* be practised, it must be evident that the previous shortening of the limb will be a considerable help to repair, as far as immobilisation is concerned. If *amputation*, the patient

has generally so far improved in condition by the previous operation, and the diminution of pain and irritation thus brought about, that he bears the operation very much better than if done in the first instance. Moreover, there can be very little doubt that the shock of removal of such a much diminished mass of tissue is *very much* less than the removal of the whole limb in one piece. Hence this all conduces to success in the ultimate issue of the case.

2. As regards the second heading, there will probably be more difference of opinion both amongst surgeons and physiologists. But very many years ago, while watching cases of amputation for diseased joints, the subsequent improvement in general health and nutrition seemed to me altogether disproportionate to the removal of the loss by suppuration. The improvement has usually been explained by the removal of the mass of diseased tissue, and thus the cessation of processes of disease (*e. g.* high temperature, &c.) inconsistent with healthy nutrition. Such, in fact, is no doubt the explanation of a great deal of the improvement, but it does not take place after excision or arthrectomy to anything like the same extent, though the removal of the diseased tissue should be in the one case as complete as in the other. Hence I was led to the belief that the mere cutting off of the limb (*i. e.* of so much healthy tissue, which *uses up* the blood more than it forms it) is a more probable explanation. With this view I have amputated through the knee in sundry cases of pure hip disease, and I believe that the improvement shown by the patient in some of the cases narrated subsequently must be attributed to this cause.

Returning to the case which forms the subject of this paper, the temperature fell to normal for a few days after the amputation, but then showed a tendency to rise, especially in the evening, so that while the morning averages were about normal, the evening varied from 100° to 102° Fahr. Nevertheless a very great improvement took place in the boy's health. He took his food better, slept better, improved in nutrition, and the sacral bedsore

healed considerably. For some time we were in hopes that the hip would quiet down and anchylose. But suppuration occurred in the stump, and abscesses were opened on August 13th and September 11th. It was for some time doubtful whether these abscesses were connected with the stump or with the hip. The subsequent history showed that they were most probably connected with the hip, and on October 7th, grating on movement being now very distinct in the joint, I excised it. I ventured to do this, believing that the gain in the lad's strength since the amputation was sufficiently great to justify it.

The improvement which followed the excision was very great. The temperature fell to normal; he steadily gained in strength and weight, and finally left the hospital on June 20th, 1880. He has remained quite well since, and has been often seen and heard of.

Reviewing the case and the possible alternative lines of treatment at the different stages, *amputation of the hip-joint* when the patient was first seen, which many surgeons would have considered the only justifiable line of treatment, would most likely have proved fatal in his then extremely precarious condition. Moreover, even if successful, it would have left the patient without a good stump to fix an artificial leg to. *Excision of the hip* was of course out of the question while the mass of tubercular necrosed bone remained about the leg. Secondly, after the leg had been amputated successfully, amputation at the hip (instead of excision) would again have left the patient with a shorter and inferior stump to that which he at present possesses.

The advantage, therefore, to the patient of this line of treatment is that he now has a stump, upon which he can wear an artificial leg of very much greater utility than anything possible, supposing the limb to have been amputated at the hip, and that he is now alive and well, whereas any other line of proceeding would have immeasurably increased the risks to his life.

It is clear that this method of treatment must be appli-

cable in a much wider class of cases than the one just described. Thus, of the succeeding cases, the first is one of pulpy disease of the left knee; this was excised, and during the rather tedious convalescence pulpy disease of the *right* hip, *i. e.* of the hip on the opposite side, showed itself. The *left* leg was accordingly amputated through the lower third of the thigh, with the result that the hip quieted down, and he left the hospital with the stump healed, and with no pain on pressure over the hip. He remained at home a twelvemonth, when signs of hip-joint mischief recurred. He came into hospital again, and had the hip-joint excised; this healed and he did well. This case is believed to be an example of advantage No. 2, *i. e.* the cutting off of a mass of blood-consuming tissue, the great improvement in health thereby resulting, so that the opposite hip-joint disease became quiescent.

In the two succeeding cases (Group II) Case No. 3 is the one which forms the text of this paper. Case 2 is a very similar one. It is a case of hip-joint disease and acute necrosis of the left fibula. The leg was amputated above the knee-joint; the hip-joint quieted down after this amputation, and he left the hospital with the joint apparently firmly ankylosed. He showed himself two years after. A sinus had broken out over the hip and at times discharged rather freely, but apparently he did not find it very inconvenient, for he did not apply for treatment, and I have not seen him since. It may be a question in this case whether the amputation should not have been followed by an excision of the hip, but when he left the hospital there seemed to be no need for such an operation.

In Cases 4 and 5 (Group III) we have patients admitted for hip-joint disease, in whom excision was performed but failed to arrest the disease; amputation above the knee on the same side was consequently performed, with the result that the excised hip-joints healed and the patients convalesced.

In Cases 6, 7, and 8 (Group IV) we have patients all of whom subsequently underwent some form of amputa-

tion at the hip-joint. They are cases in which the *primary* amputation at the hip-joint would have been inapplicable, either from the likelihood of the amputation terminating fatally, or from the case not appearing at first sufficiently serious to warrant its performance. Of the three patients in this list Case 6 comes into the second category; Cases 7 and 8 come into the first. Thus Case 6 was admitted for hip-joint disease, which was excised. Unfortunately she developed scarlet fever shortly after the operation, and had it severely; this damaged the healing of the operation wound, and some caries resulted. She steadily lost ground and became excessively emaciated and weak, the limb itself becoming perfectly useless. As amputation at the hip would probably at this time have proved fatal, a Stokes-Gritti amputation at the knee was performed. She now grew much stronger, but the hip remaining useless and the sinuses not healing, amputation at the hip was performed with perfectly satisfactory results. Case 7 was a young man with phthisis, melancholia, and tubercular disease of the hip. He was in an excessively emaciated and anæmic state, and amputation at the hip would have been almost certainly fatal. A Stokes-Gritti amputation at the knee was therefore first performed. His health now greatly improved, but the hip disease remained active. His general condition appearing to preclude excision, the remainder of the limb was removed at the hip-joint. This entirely healed, and he went home greatly improved in general health. For a time also his mental condition was much more satisfactory, but he gradually relapsed in this respect, and when I last heard of him he was in an asylum, his hip remaining healed. Case 8 was a man, aged twenty-nine, who had developed hip-joint disease at five years of age. For this he was under treatment for two to three years. Subsequently he appeared to get well enough to get about upon it, though the hip-joint was probably permanently damaged at this time. About 1880, and for various years subsequently, abscesses formed

about the hip, and for fifteen months before admission he had been entirely confined to bed. On admission there was probable lardaceous disease of both liver and kidney (*i. e.* the liver was much enlarged, and the urine was very albuminous); there were also many sinuses about the hip and rectum. The limb was first amputated just above the knee, and afterwards at the hip, but the head of the femur was too firmly ankylosed to the pelvis for complete removal. He improved greatly after the amputations; the liver returned apparently to its normal size, and the albumen disappeared from the urine. After leaving the hospital he was well enough to return to work for a while, but the disease about the os innominatum was too extensive for perfect healing, and sinuses still persisted, for which he has lately again come under treatment.

Cases 9, 10, and 11 were patients in whom a fatal ending occurred. Excision or amputation through the thigh was done in all; amputation through the hip in two. All the operative procedures were carried out in safety. Death resulted in all from previous lardaceous or general tubercular disease.

It will thus be seen that this principle of treatment is applicable in a rather wide class of cases, and it is probable that further experience will enlarge the number. For example, it will probably prove applicable in multiple joint disease (mostly of a tubercular nature), affecting joints of the upper extremities as well as of the lower. All surgeons know how unsatisfactory such cases are to treat, and how often they terminate fatally, either from general tubercular or from lardaceous disease. It will probably be found that the sacrifice of a portion of one of these affected limbs so far improves the general health, that the other affected joints will become amenable to surgical treatment.

The following eleven cases of amputation near the knee in patients suffering from hip disease have all occurred in my wards at Guy's Hospital.

They are arranged in five groups.

Group I.

1. *Pulpy disease of left knee; excision; disease of right hip; amputation through left thigh; recovery; subsequent relapse; excision of right hip.*—A. W., æt. 5 (Dorcas), was admitted October 13th, 1878, with pulpy disease of left knee. It was excised on December 13th, and the after-progress was tedious. On February 24th, 1879, signs of disease in the right hip were noticed for the first time, and the knee wound was still discharging considerably. Amputation through the left thigh was performed on March 22nd. There was still pain in the right hip on the 31st, but by April 23rd it had vanished. On May 31st the stump was healed, and pressure over the right hip caused no pain, and the health was much improved. He left the hospital on August 1st.

He was readmitted on June 15th, 1880, for a relapse of the disease in the right hip. This was excised and he did well.

Group II.

2. *Destruction of the left hip; acute necrosis of left fibula; amputation above knee-joint; improvement of hip disease; ankylosis; subsequent sinus.*—A. P., æt. 14 (Naaman), was admitted April 13th, 1879, with an acute illness of two days' duration. Active delirium; temp. 104°. The left hip was evidently acutely diseased. Bryant's line on the left side measured $1\frac{5}{8}$ inches, and on the right $2\frac{5}{8}$ inches; and the left trochanter was much thickened. On May 4th the left leg was found swollen, and fluctuation was detected. It was incised, and the whole fibula found bare. On June 16th the patient was much wasted, and there was still much pain in the hip and knee. On the 22nd Gritti's amputation was performed at the knee-joint. The hip trouble quickly subsided, and on September 20th it is noted that the hip was firmly ankylosed. On November 21st he was discharged on crutches with no pain in the hip.

He came to the hospital on March 27th, 1881. "A sinus connected with the hip had appeared some time after he went home, and at times discharged freely."

3. *Acute necrosis of tibia; amputation above knee; acute suppurative hip disease; excision; recovery.*—W. W., æt. 15, was seized suddenly in April, 1879, with acute pain in the left leg; matter formed and was let out, and he was admitted on July 25th with the left tibia and ankle-joint far advanced in a condition of necrosis. There was no albuminuria; the liver was large, and he had bedsores. There was also some swelling about the left hip-joint, which was thought to be pulpy,

but the general condition was so bad that it was difficult to make out the exact state of the joint. Stokes-Gritti's amputation was performed on the 26th. On August 2nd the stump was doing well, but the temperature rose to above 100° and acute suppurative disease of the hip (left) set in, abscesses being opened on August 13th and September 11th. On October 7th the hip was excised and much pulpy material removed. From this time the boy's health rapidly improved.

He left the hospital on June 20th, 1880, and remains well to the present time. He is often heard of.

Group III.

4. *Left hip disease; strumous manifestations in other joints and fingers; excision; Gritti's amputation; convalescence.*—S. S., æt. 7 (Dorcas), was admitted in August, 1879, with disease of the left hip of six months' duration. There was thickening of the tissues around the metacarpals of both right and left hands, and around the right elbow. There was some effusion into the right knee. Sinuses were freely laid open on October 7th. The hip was excised on November 14th. After this the notes were very inadequate; but on March 9th, 1880, the left leg was removed by Stokes-Gritti amputation. Then the patient improved greatly in health; the sinuses quickly granulated up, and on May 8th she was discharged with instructions to return to hospital if she did not get quite well. There is no note of her having been up to hospital since.

5. *Suppurative hip disease; excision; progressive emaciation; Gritti's amputation; convalescence.*—F. G., æt. 15 (Naaman). Disease of the left hip began in January, 1881. It was treated by ordinary methods till September, 1882. On the 18th October, 1882, he was admitted into Guy's, when suppuration had occurred. On October 20th the abscess was opened and the hip excised. On December 12th another abscess opened spontaneously below Poupart's ligament. Though the sinuses discharged but little the patient failed to improve, and remained much emaciated. On December 20th Gritti's amputation at the knee was performed. On January 1st his general condition was much improved. On the 16th the stump was healed, and he continued to gain flesh and strength. He was discharged on March 7th, on crutches, and the sinuses were noted as being very nearly healed. There had been no albuminuria.

Group IV.

6. *Left hip disease; excision; progressive advance of disease for three and a half years; Gritti's amputation; amputation at the hip six weeks later; spinal trouble; recovery.*—N. S., æt. 22 (Charity), was admitted March 19th, 1879, for left hip disease. Excision was performed in October. She developed scarlatina within the first fortnight, and this damaged the healing of the wound, so that some necrosis occurred about the os innominatum, and small pieces of bone came away. She went home, but in March, 1881, she was readmitted, and sinuses about the hip were explored and scraped out. From this time till 1883 she had to lie on a couch, and bone continued to come away.

She was readmitted January 22nd, 1883, with the left leg pendulous, inverted, shortened, and useless, with scars that were healed over but still tender, and with loss of sensibility below the knee. On February 2nd Stokes-Gritti's amputation was performed to improve the patient's general condition, and shorten the lever of the useless leg. After the operation the hip was easier, but the stump gaped when the sutures were removed, and was not healed till April 13th. She then was able to sit up. Subsequently she suffered great pain in the hip, and on May 29th Furneaux-Jordan's amputation was performed, She was discharged quite healed on July 14th.

She was readmitted for a doubtful spinal condition on April 23rd, 1884. She was greatly improved in condition, being plump and well nourished. There was occasionally a little discharge from the inner side of the stump. For this she was ordered to wear a Sayre's jacket. She has continued to wear these and poroplastic jackets ever since (the plaster jacket, however, giving the greatest comfort). With this support she is able to get about fairly actively and perform ordinary household duties. She writes, February, 1892, that the hip is quite well, but her spine gives her a good deal of trouble.

7. *Phthisis; left hip disease; suppuration; incision; amputation through lower third of thigh; amputation at hip; recovery.*—L. P., æt. 28 (Naaman). In September, 1880, Dr. Douglas Powell diagnosed phthisis.

On February 15th, 1886, he was admitted into Guy's. He then had early disease of the left hip, and was kept in a Bryant's splint four months. He was admitted to Naaman on May 29th. In October abscess had formed, and there was half an inch shortening. Amputation through the lower third of the femur was performed on November 2nd, and the abscess over the hip opened. The stump healed by November 24th, but there was a good deal of discharge from the hip, and on December 7th

the limb was amputated at the hip. The stump healed well, but another abscess formed and was opened, and eventually he was discharged to a convalescent home on July 2nd, 1887, with the stump quite healed, and all the sinuses closed. This patient had been melancholic for some time before admission. He remained so throughout his stay in the hospital, but improved greatly after the operations towards the end of the time. He continued to improve after his return home, and was able for some time to return to work; but he finally relapsed, and was last heard of in a lunatic asylum, though the stump remains healed.

8. *Right hip disease; prolonged suppuration; phthisis; lardaceous disease; rectal fistula; amputation through thigh; tedious after-progress; removal of rest of femur; convalescence.*—E. H., æt. 29 (Naaman). Hip trouble began when he was five, after a fall from a perambulator. Extension was applied for eight months in St. Thomas's Hospital, and he was subsequently manipulated by Hutton the bone-setter. He got about quite well till 1880, and in 1880, 1886, 1887, and 1888, various abscesses formed about the right hip, and were opened. He had been in bed fifteen months before admission to Guy's on September 20th, 1889.

The right hip was then dislocated and ankylosed on the dorsum ilii; there were discharging sinuses and a rectal fistula. The liver and spleen were enlarged, and the urine contained 1·05 grammes per litre of albumen. There was old phthisis at the left apex, and recent at the right, and the temperature was hectic.

On October 1st the thigh was amputated through the lower third. The progress after this operation was tedious and anxious. There was frequent secondary hæmorrhage, owing to an exceedingly fœtid abscess having been cut across in the amputation. This poisoned the stump in spite of all antiseptics, and produced septic osteo-myelitis. The end of the femur necrosed and protruded from the stump. Two inches more of the bone had to be removed, and the wound eventually to be plugged and dressed under $CHCl_3$.

By October 28th he was gaining ground.

On November 1st the rectal fistula was operated upon, and on November 11th he was sitting up. From this time till March 28th, 1890, there were several minor operations upon both hip and rectal sinuses. The liver diminished in size.

On May 9th the shaft of the femur was found in a diseased state, and was dissected out. An old sinus near Poupart's ligament broke out on August 6th, but on the 22nd the wounds were granulating and there was no discharge. On the 23rd he was discharged to a convalescent home.

Throughout the case albumen was rarely absent from the urine; from 1 in 1000 it diminished to a "trace," a "slight trace," and a "very slight trace," and the diminution in the size of the liver was very marked.

GROUP V.

9. *Left hip disease; sinuses; excision; lardaceous disease; amputation through thigh; amputation at hip; death from lardaceous disease.*—M. S. When three years old disease of the left hip began after a fall. She was treated at St. Thomas's in 1871, and in 1872 at Guy's, with rest. Suppuration occurred, and she was readmitted to Guy's, and the abscess opened and the separated epiphysis removed on October 11th, 1872. She remained well till 1885, when there was a relapse following injury. The trochanter and neck were excised. After leaving the hospital the disease was aggravated by another fall. She was readmitted in November, 1885, and had then excessive lardaceous disease of viscera. The urine became nearly solid on boiling. Amputation through the lower third of the femur was performed to shorten the lever. The stump healed, and the patient left the hospital on February 3rd, 1886.

She was readmitted in March, the discharge from the hip becoming more troublesome. The lardaceous disease was unaltered, but the patient's general condition was improved. On May 7th the limb was amputated at the hip and the acetabulum found bare. She did well till six days before she died, and the stump healed, but on May 28th she began to have pain in the back, was sick, and refused to take food; became sleepy and hardly took notice of any one, and died on June 1st.

Post-mortem.—There were scattered tubercles at each apex. Acute pericarditis. Very extensive progressive disease of the pelvis and extensive lardaceous disease of the spleen, kidney, liver, and intestines.

10. *Left hip disease; albuminuria; amputation through thigh; excision; amputation at hip; prolonged suppuration; death.*—W. W., æt. 17. When two years old he attended at Guy's for hip trouble following a fall, and was discharged "cured." He went away till April, 1880, and could flex the joint to a right angle. In April an abscess formed in the groin, and he left work. He was admitted on April 27th, 1880, with active disease of the left hip, with dislocation of the head on to the dorsum ilii, two copious discharging sinuses, and slight albuminuria.

On September 10th amputation through the thigh was performed, and

a large abscess opened over the hip. On October 5th a hip abscess was again incised, and the joint found partly ankylosed. Excision was performed on October 26th, and the finger passed into the pelvis in two places. Notwithstanding the profuse discharge, occasional diarrhœa, and albuminuria, the patient gained flesh fast. On January 4th, 1881, amputation at the hip was performed, and the stump healed fairly well. There was still much discharge, yet he improved, and in May was getting up. During the summer his condition fluctuated, but the profuse discharge and a hectic temperature gradually reduced his strength, and eventually he sank into a comatose state, and died on September 13th.

No post-mortem report.

11. *Right hip disease with sinus; amputation through thigh; excision of hip; lardaceous disease; old and recent tuberculosis; death.*—S. S., æt. 44, an old soldier, was admitted on October 2nd, 1887, with the right knee acutely flexed and the hamstring muscles contracted. The right hip was flexed and fixed, and there were marks of old sinuses. Three times under ether forcible extension was made and the knee straightened.

There was occasional discharge from the hip sinus. In January and February unhealthy sores formed along the leg, probably due to the splint; the limb became very painful and the patient's condition bad.

On February 27th the leg was amputated above the knee, as he was not in a state to stand amputation at the hip. On March 10th the wound was nearly healed, and the hip was excised on the 23rd and found full of pus. This was followed by profuse discharge, bedsores, and sweats. The urine contained one ninth of albumen. In May necrosis of the sacrum from a bedsore occurred. In June he grew weaker, and the abdomen became distended, and death took place on the 15th.

Post-mortem.—There was extensive tubercular disease of femur and pelvis; tubercular peritonitis and old tubercle of the lung; lardaceous disease of the kidneys, liver, spleen, and intestines.

In Group I the amputation and the hip disease were on different sides. The result is an illustration of the way in which, in tubercular disease of more than one joint, the general health may be so greatly benefited by the removal of one exhausting focus that the remaining disease may go on to cure.

This case may be compared with the two cases in the next group in which the limb was shortened on the affected side *before* excision of the diseased hip was performed. In the first of these two cases (Case 2) the hip ankylosed after the excision, and though he showed himself subsequently at the hospital with a discharging sinus, he did not ask for any

subsequent treatment. In the second case (Case 3) subsequent excision became necessary, and the most satisfactory results were obtained.

There can hardly be a doubt that the facilities which the shortening of the limb gave for a less irksome and confining treatment contributed considerably to the satisfactory results.

Group III includes Cases 4 and 5. Excision was first performed in both cases. In one after two months' interval the patient remained emaciated and unimproved; in the other after four months the necessity for further operative treatment was held to exist, but the patient's state cannot be described in consequence of the absence of notes at this period. In both cases Stokes-Gritti's amputation was performed, and was followed by greatly improved health and convalescence, which, as neither case has reappeared, it is possible to hope has been permanent.

Group IV contains three cases in which the whole limb was removed by instalments. In Case 6 the disease was severe and of long standing. In Case 7 the disease was rapid and the patient was phthisical; and Case 8, with phthisis, lardaceous disease, and rectal fistulæ, was an unusually bad and unpromising case. But two left the hospital to all appearances cured, and the third convalescent.

Group V includes three fatal cases. In all there was extensive disease of the pelvis and lardaceous disease. The operative procedures were carried out in safety and did well, but death resulted from the lardaceous disease in two instances, and in the third from lardaceous disease and other tubercular lesions.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 15.)

TWO CASES

ILLUSTRATING THE

TREATMENT OF ADVANCED HIP-JOINT DISEASE

BY MR. HOWSE'S METHOD OF PRELIMINARY AMPUTATION AT THE KNEE.

BY

R. LAWFORD KNAGGS, M.C.

(COMMUNICATED BY MR. HOWSE.)

Received April 12th—Read November 8th, 1892.

THE method of treatment in advanced hip disease by preliminary amputation near the knee, which the following cases are intended to illustrate, I first saw carried out at Guy's Hospital by Mr. Howse.

To that gentleman, I believe, is due the credit of conceiving and applying a plan which, though giving admirable results in practice, seems to be universally regarded with incredulity and amusement by those who hear of it for the first time.

Where excision has proved useless, or where amputation at the hip would be fatal in consequence of advanced

hip mischief being associated with general feebleness and visceral disease, the method suggested by Mr. Howse will enable a portion of a limb to be retained, or a life, that would inevitably have been lost, to be saved.

This plan is to remove the limb by instalments.

The first step is amputation near the knee.

After this the improvement in health is rapid and considerable. The failing powers are helped, because (1) the amount of the impoverished limb requiring nourishment is diminished; and (2) there is greater freedom from pain in consequence of the shorter leverage of the limb.

As soon as the wound is firmly healed the patients are able to get up and go out of doors on crutches, and after an interval of two or three months their general state is so much improved that removal of the shortened limb at the hip-joint involves infinitely less shock and risk than if amputation at the hip had been carried out as a primary measure.[1]

Case 1.—Eliza M—, æt. 8½, when first seen on July 22nd, 1887, was already the subject of advanced disease in the left hip. There was two inches shortening, and dislocation on to the dorsum ilii seemed to be present. An abscess which extended under Poupart's ligament, and was limited at the brim of the pelvis by the iliac fascia, was opened in front of the great trochanter and drained. The urine contained no albumen; sp. gr. 1024.

On October 8th the discharge from the abscess was still considerable; the liver was enlarged, reaching 2 inches below the costal margin; and the urine contained one fifth of albumen and hyaline casts. Sp. gr. 1016. On October 20th there was only a trace of albumen, and on November 6th it was absent. The child had improved, and the discharge from the wound had diminished. At the end of December the liver reached as low as the

[1] These cases were under the care of my father in the Huddersfield Infirmary, and by his kind permission I am enabled to make use of them.

umbilicus, and bare bone was felt in the acetabulum. In January, 1888, the child grew worse, the discharge increased, and the albumen reappeared. On March 9th, 1888, the patient's state was so unsatisfactory that it was generally agreed that to amputate at the hip or to leave the case alone must inevitably end in death.

It was decided to adopt Mr. Howse's plan. Amputation at the knee was performed, and in spite of an attack of erysipelas, and the formation of an abscess in the front of the stump, the child had quite recovered from the operation by the middle of May. At that time the liver was smaller, the discharge less, but a trace of albumen persisted.

On July 10th she had been out of hospital three weeks with the sinus closed. The albuminuria had disappeared. On August 27th the sinus continued closed, but there was slight tenderness on pressure over the cicatrix and above Poupart's ligament. She looked fat and well but pallid, and weighed 4 st. 2 lbs.

In September she caught cold, the sinus reopened, and she began to lose flesh rapidly. The urine remained free from albumen.

On October 19th Furneaux-Jordan's amputation at the hip was performed. The head was not dislocated, but had penetrated the floor of the acetabulum; and the shaft of the femur, which was no thicker than an index finger, was broken in attempting to lever out the head. When the head had been dissected out the opening in the acetabulum led into a cavity lined by granulation tissue, in which small fragments of bone could be felt.

The opening was enlarged and the cavity scraped; still portions of bone were felt, but the patient was suffering from considerable shock, so the cavity was plugged and the operation concluded.

There had been much oozing from the small vessels, especially in the periosteum, but the femoral artery was about the size of the radial.

The future progress was slow and uneventful; portions

of bone came away from time to time, and in the middle of March, 1889, she was sent to a convalescent home at Southport. She returned in May in good health but pasty-looking. There was no tenderness about the iliac or inguinal region, but there were two discharging sinuses. The liver could be felt 1½ inches below the costal margin, and there was slight albuminuria. Her weight was 3 st. 11 lbs.

From this time she was frequently seen about with a crutch, looking well. She was examined on January 23rd, 1892. Both sinuses had been completely closed for a year, and there was no tenderness anywhere. The liver could not be felt below the costal margin. She was growing a big girl for her age, muscular and well conditioned. Weight 5 st. 5 lbs. She carried plenty of colour, and bore only a slight resemblance to the invalid of two years ago.

A very distinct trace of albumen, however, was still present in the urine.

Case 2.—Ethel B—, æt. 9, had had the left hip excised eighteen months previously for disease of a year's duration.

On March 9th, 1888, a sinus, which had never closed, was found to lead into the pelvis, and from it a good deal of pus discharged. There was no albumen in the urine and no sign of lardaceous disease.

The child's general condition was so unsatisfactory that amputation at the hip seemed to offer the only chance of recovery, and yet it seemed hardly probable that she would survive the shock of that operation.

It was determined, therefore, to adopt Mr. Howse's plan, and the left leg was amputated at the knee-joint. An excellent recovery followed, and on April 10th she was going about on crutches—the hip being kept at rest in a Thomas's splint.

The discharge from the hip diminished considerably. Before she left the hospital for the convalescent home she

was quite a different being. She had gained a great deal of flesh and colour, and spent the greater part of the day in running about the garden on her crutches, always ready to bear witness to the improvement in her health and comfort resulting from the operation.

In August, 1888, the child was readmitted not looking so well, and the discharge from the sinus was increasing.

On August 10th Furneaux-Jordan's amputation was performed. The sinus led to an opening in the acetabulum. This was enlarged and loose bone felt within the pelvis. By syringing, a piece of green, necrosed, foul bone about as big as a sixpence was got away with some decomposing blood-clot and pieces of granulation tissue. The old sinus was enlarged and scraped, and the pelvic cavity plugged through this sinus. The cavity was large enough to permit of exploration by the little finger introduced to the second joint. The shaft of the femur was about the thickness of the little finger. It was very difficult to dissect out from its firm adhesions to the acetabulum. The femoral artery was not larger than a good-sized radial artery, and there was not much bleeding. The shock was severe.

She made a satisfactory recovery, and went out with the stump quite healed on December 1st, 1888. She returned in a few weeks, a fresh abscess having opened in the groin. In March, 1889, she was sent to Southport, and on July 4th, 1889, there were two sinuses, one in the groin, and one in the track of the drainage-tube leading towards the seat of the original mischief, and discharging a good deal of matter. Weight 3 st. 8 lbs. She had lost a good deal of flesh and was looking decidedly worse, and the urine contained albumen, uric acid crystals, and hyaline casts. The liver was not enlarged. Two years later she was again seen (July, 1891). She was then looking very well. The sinuses still discharged, and the urine contained more than a trace of albumen. Her weight was 4 st. 7 lbs.

These cases may be considered to have put the merits of Mr. Howse's plan to a fair test.

Eliza M— had been the subject of lardaceous disease for some months, during which amputation at the hip had been twice rejected at full consultations of the staff as too hazardous.

For Ethel B— nothing but amputation at the hip seemed to offer a prospect of recovery, and the risk of a fatal result from shock was thought to be more than usually great. Any attempt to deal thoroughly with the acetabular mischief would have been almost sure to have turned the scale against her. The leg was removed in each case on the same day, and except for a considerable amount of shock at the subsequent Furneaux-Jordan operation in each instance, neither child gave any real cause for anxiety.

Both of them readily acknowledged their increased comfort after amputation at the knee in almost identical terms—"because it pains me less," and it is not difficult to understand the relief they must have felt at the restraint of a Bryant's double splint being replaced by a stump which they could manage for themselves when it was dressed.

When the stumps, after the preliminary amputations, were healed, both children were able to get up and go about on crutches, the hip being fixed in a Thomas's splint. They spent the greater part of the day in the open air, and were continually on the move. The effect of the fresh air and the absence of pain were very marked, and both children gained flesh rapidly. But the manner in which the hip mischief was influenced was even more remarkable.

In Ethel B— the amount of discharge diminished considerably, but in Eliza M—, the subject of lardaceous disease, the discharge gradually ceased, the albuminuria disappeared, and the sinus healed; and, except for a little tenderness on pressure for a period of three months, the last two of which were spent in most unsatisfactory

surroundings, there was nothing to show that there was any active mischief going on in the hip at all.

In the amputations at the hip the result of the previous shortening of the limb, and of disuse, was evident in the greatly diminished size of the femoral artery and in the atrophied state of the femur. In each instance this led to fracture in the manipulations to dissect out the head.

The chief trouble was hæmorrhage, not from the femoral artery which was too small to cause anxiety, but from the numerous small vessels that were cut, and from the general oozing that took place. When the femur was being removed from its enveloping muscles, and the adhesions between the neck and the acetabulum were being divided, unfortunately the bloodless method was not very useful, because the elastic bandage had to be fixed round bony portions of the pelvis, and the supply of blood through the apertures in the innominate bone could not be satisfactorily controlled without hampering the operator.

In cases where the pelvis is implicated, as in both of these, the removal of the limb by instalments offers great advantages.

The less serious character of the final amputation, and the improved condition of the patient when it is undertaken, justify the operator in more careful and thorough investigation of the pelvis, and in the adoption of more radical measures for the removal of disease than in similar cases, where amputation at the hip is performed in the orthodox way.

The shock of this operation, always great in a child much reduced by pain, prolonged suppuration and confinement in bed, would be largely increased by extending the operation sufficiently to deal thoroughly with the disease within the pelvis.

In conclusion, it may be stated that Mr. Howse's method does not aim at obtaining results superior to those

gained by primary amputation at the hip, but at reaching the same end in a very much safer way.[1]

[1] This was written two or three years ago. The increased acquaintance with the merits of this method of treatment that I have gained by assisting Mr. Howse in the collection of his cases would lead me to modify this last statement. The results are in some cases decidedly superior to those obtained by amputation at the hip-joint (*vide* Mr. Howse's cases).

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 15.)

BURSTING OF A LARGE HEPATIC ABSCESS

INTO THE

PERITONEAL SAC;

INCISION OF THIS LATTER; EVACUATION OF PUS; FLUSHING; RECOVERY.

BY

J. W. HULKE, F.R.S.

Received November 9th—Read November 22nd, 1892.

THE bursting of a large abscess into the peritoneal sac is so usually followed by rapidly fatal peritonitis, that an instance of recovery after this accident has seemed to me of sufficient interest to offer to the notice of the Royal Medical and Chirurgical Society.

In June, 1891, with Dr. J. L. Paul, I saw a gentleman, æt. 33 years, who three years previously had been invalided from India, having there had, he told us, "liver disorder which had left his health very broken." Upon his return to England his health improved so quickly that after a few months he regarded himself as quite well, and he resumed his employment—mercantile. In March, 1891, symptoms of hepatitis again supervened; and soon after this such a copious expectoration of pus

occurred, as left little room to doubt that an hepatic abscess had burst through the diaphragm, and emptied itself by the lung. Upon this, his condition again improved so that he was again able to take up his official work, though he remained far from strong; and in June he was again laid up with "liver disorder."

On 9th June (1891), when, with Dr. Paul, I first saw him, his symptoms were—"stitch in the right sixth intercostal space, and great tenderness there in a small spot intersected by a sagittal line drawn through the anterior fold of the armpit. The upper limit of hepatic dulness distinctly higher than normal. He had fever, he was haggard, and he looked and felt seriously ill.

Read in the light of his previous history, these symptoms appeared to be conclusive evidence that another abscess had formed in the liver, and also to hint that unless anticipated by timely incision, this would probably break into the lung, as the first one appeared to have done in the preceding March—an event it was most desirable to avoid.

Since, however, an incision made through the tender spot in the sixth intercostal space, into the liver, would have crossed the lower angle of the pleural sac, the diaphragm, and possibly the lower margin of the lung; and since from the absence of "fulness" in that space, and also of superficial œdema there (so frequently present over a deeply-seated abscess), it appeared not unlikely that the structures named might be already consolidated and agglutinated in such a way as to bar off the line of the wound from the pleural and the peritoneal sac, it was deemed by us prudent to defer the operation for a short time, the patient being in the interval closely watched by Dr. Paul, with a view to the prompt evacuation of the abscess by incision so soon as we had clearer indication of the course it was taking.

Two days later, sudden, very profound collapse supervened, the temperature fell below 97° F., and the patient appeared to be dying. Twenty-four hours later the col-

lapse was a little less extreme, and when I saw him on the second day, with Dr. Paul, he had slightly rallied.

The upper limit of hepatic dulness was then distinctly lower than before; but from the costal cartilages downwards, nearly as low as Poupart's ligament, and from two finger-breadths to the mesial side of the right *linea semilunaris* outwards to the flank, the right side of the belly had lost its normal resonance and become dull.

Under these circumstances it could not be doubted that the hepatic abscess had burst into the peritoneal sac. Incision into this latter and flushing out appeared to offer the only chance of rescue. Chloroform was given. The abdominal walls were divided by an incision parallel to, and just external to the *linea semilunaris*. The peritoneum, subserous fat, and fascia transversa were not separately distinguishable, owing to œdema and infiltration. A large quantity of pus escaped from the peritoneal sac when this was opened. Of this, about Oj was caught in a vessel, and much more escaped into the bed and could not be measured. The cavity was then flushed out with a warm solution of boric acid in water until this returned quite clear and colourless. Next a large drain-tube was inserted, the ends of the tegumental incision were brought together, and an absorbent antiseptic dressing was applied.

The space containing the pus was bounded anteriorly by the parietal peritoneum, posteriorly by the convex upper surface of the liver above, and below by the intestines, coils of which were plainly recognised with the finger.

From the time of the operation the patient's desperate condition began to improve. Two days later the tube was shortened, as it seemed as if it were being pushed out by lessening of the cavity. He continued to make progress, and at the end of three weeks, the daily amount of discharge having greatly lessened, a smaller tube was substituted for the large one. At the beginning of July he was able to leave his bed and to sit up, and at the middle of that month he returned to his home in one of the suburbs.

After this he had a recurrence of pain and fever, attributed to premature withdrawal of the drain-tube. This was with some difficulty re-introduced by Dr. Paul, and the unfavorable symptoms disappeared. A tube was worn until November, when the wound closed.

The recovery of the patient without prompt evacuation of the pus from the peritoneal sac would, it is suggested, have been a most improbable circumstance, and it is claimed that his rescue may reasonably be ascribed to the incision and flushing. The success which attended these measures is an encouragement for the adoption of the like practice in similar circumstances.

It appears probable that during the extreme shock immediately supervening on the rupture of the abscess into the peritoneal sac, wider diffusion of the escaping pus was hindered by agglutination together of the intestines, thus confining it to the right side of the belly. How perilously weak was this barrier was apparent from the distinctness to touch of the coils of gut noticed at the operation; they seemed to float in the pus.

The spot where the abscess broke through the liver-capsule was not ascertained, but it could not be doubted that it was in the upper surface of the liver; its discovery had not any bearing on the technique of the operation, whilst the delay, and the disturbance of parts, entailed by much searching for it, would have added much to the patient's already very great peril.

The line of treatment here practised has been pursued in certain instances of peritonitis, *e.g.* those due to tuberculosis and to mechanical injury, with such a degree of success that it may fairly claim wider adoption. The idea is not novel: my co-librarian, Dr. Gee, has reminded me that it was long since suggested by John Hunter. For my own part, with present experience, I recall with regret recollections of fatal cases in former years in which a bolder treatment might have had a different issue.

Addendum.—Since the above notes were written the

patient has had a relapse, and his condition is now more unsatisfactory. In April of this year (1892) he had a slight rise of temperature, and with it uneasiness which he referred to the region of the liver. No enlargement of this organ was then perceptible, and these symptoms soon disappeared, but in the latter part of July he had again fever, and the liver was now found enlarged. There was also now tenderness in a very limited area at the right side of and slightly below the ensiform cartilage. He entered the Middlesex Hospital, where, on August 2nd, by an incision through the tender spot, an abscess was opened and emptied. This incision, from its direction and the depth at which the pus was reached, was believed to have entered the liver itself. Immediate relief was afforded, and on the 20th of that same month he went home, with the injunction to wear the tube until all or nearly all discharge had ceased. He gained flesh and strength, and returned to office work finding little inconvenience from the presence of the tube, which he had learned to manage. Except for this at the beginning of the present month he appeared quite well. All sign of enlargement of liver had disappeared. Quite recently, however, he has had a slight feverishness which made him apprehensive that an abscess is again threatening, although the region of the liver was free from tenderness, pain, and swelling. As, however, coincidently with the feverishness he had painful swelling of the knee-joint, and he had had, some years before, acute articular rheumatism, it seemed likely that the fever might be symptomatic of gonitis, and this the sequel established, since the rise of temperature disappeared after a few days with the recovery of the knee.[1]

The danger attending the bursting of an hepatic abscess into the chest, and its discharging through the lung is well illustrated by the following case; for, although no necropsy could be obtained in verification of

[1] The patient, I am informed, is now apparently quite well.—J. W. H., July 19th, 1893.

the diagnosis, the clinical signs clearly indicated the liver to have been the focus of the abscess.

In February, 1876, Captain —, æt. 35, on service in India, had an acute illness regarded, he told us, as pleurisy; and a few weeks later, after an imperfect recovery, he had a second similar attack, which left his health so greatly impaired that he was invalided home to England. During his voyage his illness increased so greatly that it was scarcely expected that he would reach this country alive.

On the 29th May (of the same year) I saw him in consultation with the late Dr. Ch. Murchison and his medical attendant Dr. Thorne. His emaciation and weakness were extreme. His hands and feet were puffy. His urine was free from albumen. The right side of the chest was dull as high as the collar-bone. The intercostal spaces posteriorly below the lower angle of the shoulder-blade, and also laterally in the mid-axillary line, appeared to slightly bulge. Vocal thrill was scarcely perceptible. The heart was displaced much to the left of its normal position. The area of hepatic dulness was prolonged downwards to below the level of the navel; and throughout this large dull area there was distinct tenderness. He had extreme dyspnœa, temporarily and slightly relieved by coughing up large quantities of pus.

Dr. Murchison's diagnosis based on the above symptoms was hepatic abscess which had burst into the chest and lung. It was decided to verify the presence of pus in the lung by aspirating, and, if found, to evacuate it by incision and to drain, should this under the circumstances ascertained at the time be practicable. Nitrous oxide followed by ether was given by Mr. Braine. A long aspirator trocar was pushed into the chest one hand's-breadth below the lower angle of the scapula, and at a distance of six inches outwards from the vertebral spinous processes, and when the trocar had reached a depth of

3¼ to 3½ inches pus began to flow. One pint of very viscid, rusty, inodorous pus was drawn off. The patient's exhaustion was now so extreme that it was judged prudent, in the imminent risk of his death, to defer the longer measure of incision and drainage for a couple of days in the hope that, rallying, he might be better able to bear it. Two days afterwards, dyspnœa having again become very urgent, his chest was again aspirated, in my absence from home, by another surgeon. The result was negative, pus was not found, and a few hours later death ensued. A necropsy was not permitted. That the pus here drawn off was not in the pleural sac but contained in a large abscess-cavity in part within the lung itself is, I submit, a fair inference from the great depth to which the trocar passed before pus began to flow. Obviously if the diagnosis was correct the cavity could not have been perfectly drained by a thoracic incision alone; for its efficient drainage a lower incision through the belly-wall into the liver would have been necessary. The marked tenderness of the hepatic area suggested the probability of the presence of peritoneal adhesions lessening the risk of the escape into the peritoneal sac. If it be asked why, in view of these circumstances, such lower opening was not made in the first instance; the apology is that the presence of pus in the chest was already known, whilst liver-abscess had only a high degree of probability. Moreover in India, where his illness began, it was regarded as pleurisy, and since the patient's arrival in London, before Dr. Murchison saw him, we were informed that two physicians much occupied with chest-diseases had examined him and pronounced him to have empyema only, and these opinions were not wholly without influence.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 22.)

THE RADICAL TREATMENT

OF

SEVERE TALIPES EQUINO-VARUS IN CHILDREN.

BY

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HOWEVER keenly some of the views set forth in the following paper may be traversed, this statement, I think, will be allowed to pass unchallenged, that the old-fashioned or orthodox treatment of severe club-foot by subcutaneous tenotomy and the subsequent use of Scarpa's shoe, left much to be desired. For my own part I am prepared to admit that I found it entirely unsatisfactory in dealing with congenital talipes in hospital out-patient departments, and tedious, and ofttimes disappointing, in practice in the wards.

An important feature in the orthodox treatment of club-foot consisted in the subcutaneous division of the tendon of Achilles last of all. Thus, in the most recent edition of Erichsen's 'Surgery'—to which book the surgeon naturally refers when in search of authority—we read as follows:—"After these [the tendons of tibialis anticus

and posticus] have been cut across, the tendo Achillis should be divided; but it is better in all cases to delay this for some weeks, as it steadies the foot and thus facilitates the correction of the lateral displacement." But even after the Achilles tendon had eventually been divided, so much force had to be brought to bear upon the foot that the child was apt to suffer severely, and if the surgeon, or the dresser or nurse, persisted in its energetic employment, it was at the risk of producing an excoriation or even a slough upon the instep. Thus, treatment was apt to become intermittent, and progress slow and painful. Moreover, operative interference under chloroform was likely to be required time after time, and the surgeon rarely knew when the case was perfectly cured and safe from relapse.

One element in the production of this unsatisfactory state of affairs is the unreasonable prejudice which so widely existed against getting to work at the earliest possible moment upon a congenital club-foot. So the child was allowed to drift on; the tarsal bones becoming more misshapen, the fibrous structures more resisting, and the deformity more intractable.

Professor Sayre has done more than any one else to check this *laissez aller* practice, by insisting, in his own peculiar manner, that the treatment of the deformity should be delayed for no longer a period than is needed for the aforementioned attendant to wash his hands and the monthly nurse the child.

I do not think that my experience in connection with the old-fashioned treatment of congenital club-foot has been in any way peculiar, but I must honestly admit that, after operating on a child and giving him much attention in the wards, and after supplying the parents with careful instruction as to subsequent procedures, I was very apt to find that child, some months later, an unwelcome applicant for further treatment. Indeed, I am sure that my experience cannot have been peculiar, for many children who have been under the care of other surgeons,

and who have been submitted to the orthodox operative measures, have, under the guidance of blind chance, found their way to me for supplemental help. I have little doubt, moreover, that failures of my own under the old treatment have sought orthopædic salvation in other hands.

I think it far less likely, however, but still not impossible, that many children on whose congenital club-feet I have operated after Phelps's open and radical method have gone elsewhere for advice. For, after the operation has been once effectually performed, the occurrence of inversion is improbable, even though some further drawing up of the heel may take place, for the operation has less control over the position of the heel than of the anterior segment of the foot—less beneficial influence over the equinus than over the varus. (The vertical scar upon the inner border of the foot shows that the open operation has been resorted to.)

That which I have called the orthodox treatment of club-foot (as opposed to the radical method) was based upon the theory that it was inexpedient, if not dangerous, to divide a shortened tendon or band of fascia, except by means of a slender blade introduced through the smallest possible opening in the skin. But in these days of surgical cleanliness the operator prefers to know exactly what he is dividing: to have his eye or his finger always in advance of his knife. He has less dread of incising the skin than the surgeon of the past generation had of even pricking it.

However vigorously the knife may have been used in the anatomical darkness of a subcutaneous operation, the complete effacement of the deformity is effectually hindered by the skin, which is not only thick and resisting, but is closely connected with the subjacent plantar fascia. An essential feature in Phelps's operation is that the skin is freely incised, so that on the completion of the operation nothing whatever is left to oppose the complete effacement of the deformity.

Preparation.—It is hardly necessary to insist that at the time of the operation the patient should be in the best possible health; and for this purpose he may be kept under observation for a few days before operating. The day before the operation the foot is well soaked and washed. And on the morning of operation it is again washed in soap and water, and then with a 1 in 2000 solution of perchloride of mercury. After this, and till the surgeon is ready, the foot is kept enclosed in wet antiseptic cloths.

No Esmarch's band or other form of tourniquet is used. Each bleeding point is seized by the catch-forceps as it attracts attention, but, as a matter of fact, the amount of bleeding is far less than might have been expected. Perhaps one or two small vessels may require temporary pinching with the self-holding forceps, but it is quite exceptional for more than one of them to need a ligature.

The *instruments* required are a tenotomy knife for the tendon of Achilles, a medium-sized scalpel, a pair of dissecting forceps, a few pairs of catch-forceps, a pair of scissors, and a steel director. No specially designed knife is needed.

The operation.—An anæsthetic having been administered, the foot is uncovered, and the amount of resistance and deformity is estimated by forcible manipulation. The tendon of Achilles is then divided subcutaneously, the foot being flexed to the utmost and brought into the best possible position.

For many years, and quite independently of Phelps's innovation, I have been in the habit of beginning all operations for equino-varus by the division of the heel-tendon. The influence of this tendon is by no means confined to the elevation of the heel; having contracted, as it were, to the utmost in drawing up the heel, the tendon obtains a still further shortening by rotating the os calcis inwards on its antero-posterior axis. Thus, its division reduces, and in some slight cases actually effaces

each element of the congenital deformity. Such cases, however, do not enter into the class of those for which I am advocating the adoption of Phelps's operation, as they may be efficiently dealt with by the far simpler procedure. But I would like to insist on this, that in all cases of equino-varus, whether the surgeon contemplates the adoption of the subcutaneous or the open operation, he begin by dividing the Achilles tendon. After this tenotomy, the foot should be flexed with great force to a right angle, when, if a considerable amount of deformity still persist, the radical operation may be proceeded with, unless, indeed, a milder course of treatment appears to offer an equally favorable result.

The next step in the operation consists in making a free vertical incision from dorsum to sole of foot, between the tip of the internal malleolus and the tuberosity of the scaphoid. This passes across the head of the astragalus, and the top of the incision brings into view the internal saphenous vein and the tendon of the tibialis anticus. The vein is generally sacrificed, and, the foot being straightened out, the section of the tibialis anticus tendon affords considerable mechanical relief. Attention being then given to the lower part of the incision, the plantar fascia—comparatively thin as it covers the abductor hallucis—is divided, and probably a good deal of that muscle. The strong piece of the fascia over the flexor brevis digitorum is cut, and, in an extreme case, some of the short flexor itself. The tendon of the tibialis posticus is next sought in the depths of the wound, and, on its division—the foot being still further straightened out—the tendon of the flexor longus digitorum, which lies close to its outer side, may invite section. At about this period of the operation the anterior part of the internal lateral ligament may be dealt with. It is an element of the highest importance in keeping up the cramped position of the inner border of the foot, running, as it does, from the front of the internal malleolus—over the head of the astragalus—to the scaphoid bone. The old-

fashioned, subcutaneous operation entirely failed to cope with this difficulty.

After the front of the deltoid ligament has been freely traversed by the scalpel, and, by the help of more forcible manipulation, the foot has been further straightened out, the scaphoid must needs be pulled forwards from the astragalus, the astragalo-scaphoid joint being left wide open. Placing the end of the finger into this joint, the surgeon may distinctly feel the fibres of the inferior calcaneo-scaphoid ligament maintaining some deformity. When these have been divided, some of the inner fibres of the calcaneo-cuboid ligaments may be found to want a touch of the scalpel. In short, every structure must be cut, which prevents the full and unobstructed straightening of the foot.

When the bleeding has been controlled—and it is usually quite insignificant—the wound and the foot are washed over with a warm mercuric solution (1 in 4000), the yawning incision is gently stuffed, and the foot is enveloped with mercuric gauze, and the foot and ankle and the lower part of the leg are enclosed in a flannel bandage. Two lateral splints of common house-flannel are then soaked in creamy plaster of Paris, and are firmly and evenly applied under a gauze bandage. During their application and hardening the foot is held in the over-corrected position, good heed being given that the plaster casing is not dented in by any uneven pressure of thumb or fingers. The toes are left uncovered for inspection, so that early information of obstructed circulation may be obtained.

I confess that in some of my earliest operations I was apprehensive lest the anterior segment of the foot might show signs of threatening gangrene, not only because of its being rigidly confined in the gypsum casing, but because of the depth of the wound which traversed the tissues from dorsum to sole. Practice, however, has shown such fears to be groundless. The casing, though rigid, is not actually tight, and although the internal

plantar artery may perhaps be cut in the operation, the dorsalis pedis and the external plantar artery, which are of far greater anatomical importance, run no chance of being injured. But small though the internal plantar artery is, it may often be recognised in the progress of the operation and avoided. The child suffers but little distress after the operation, and on one occasion only did a house surgeon deem it advisable to remove the dressings to examine the foot. In that particular instance the toes had become blue and cold; but a fresh casing was at once applied, and everything went well. As a rule, the first dressing is done at the end of ten days or a fortnight. The casing is opened out, the bandages are cut up, and the mercuric gauze, already loosened by a little aseptic discharge, is lifted out of the wound, the surface and depths of which are thickly covered with healthy granulations. The foot is then washed and lightly dressed as before, and again secured in plaster of Paris. In six or seven weeks, or a little more, the wound is completely healed, and the treatment by manipulation and massage is entered upon.

We all know how carelessly or imperfectly instructions as regards manipulation and massage are carried out by parents when a child is under their unsupervised control; it is a severe and crucial test for the value of this radical operation that when the child is brought for inspection at the end of some weeks, or even months, the foot is found in a very excellent position. If it still shows more inversion than is desirable, it is at once seen that this defect can be corrected with the least possible application of force. Time after time I have asked the sister or nurse of a ward if the child seemed to suffer from the position in which the foot is left; and the answers which I have received have left me well assured that the change of position causes little or no distress, for the simple reason that it is kept up without any force or strain. There is no strain because every resisting band has been severed.

The deep wound which the operation leaves upon the inner aspect and in the sole of the foot at the mid-tarsal joint soon fills up with granulation tissue, which in due course becomes converted into firm scar tissue. The subsequent contraction of this new material is rarely strong enough to reproduce or to perpetuate the inversion of the foot. It does prove sufficient, however, firmly to weld the anterior with the posterior segment of the foot, and to render the corrected foot serviceable for flat standing and for progression. The sooner that the patient can be got to stand upon the flattened foot—the wound, of course, being healed—the less is the risk of even a slight amount of inversion recurring. In the progress of cicatrisation a firm fibrous splice is inserted in each one of the divided tendons and ligaments; and I apprehend that the widened space between the astragalus and scaphoid is filled in with the same material. Probably it is the interposition of this thick medium between the two segments of the foot which renders the parts so strong and trustworthy when all is healed.

In one very severe case of equino-varus, when almost every structure which one expects to encounter in the radical operation had been divided, and the foot still resisted easy adjustment in the correct form, I found that the impediment came from the tendon of the peroneus longus. Having exposed this in the depths of the sole, and having divided it, the foot at once came easily into position.

I confess that if a candidate at examination told me that the tendon of the peroneus longus was one of the structures needing division in the operation for the cure of talipes equino-varus, I should be inclined to think that his statement was based either upon a misapprehension or upon ignorance. But, as a matter of fact, this tendon may have a great deal to do with the perpetuation of the congenital deformity:—when a foot has long been cramped with an inverted sole, an elevated heel, and a contracted internal border, this tendon may

very likely be found at fault. And the greater the previous elevation of the heel the greater may be the need, after dividing the tendon of Achilles and flexing the foot, of severing the tendon of the peroneus longus. The peroneus longus is an important extensor of the foot, and when, after Achilles-tenotomy, the deformed foot is forcibly flexed, the peroneal tendon is tightened up, and is holding the base of the first metatarsal bone as closely as it can towards the heel. In other words, it is dragging the scaphoid against the head of the astragalus. And as it is the surgeon's object in performing the open operation to insert a thick wedge of space between the astragalus and the scaphoid, and to be able to maintain that space without the exertion of any considerable force, the tendon of the peroneus longus may need to be sacrificed.

I have not directed attention to this anatomical detail with the idea of suggesting that in the performance of the open operation the surgeon must expect to be called upon to divide this particular tendon, but rather with the view of emphasising two statements: first, that a surgeon cannot expect to deal effectually by a subcutaneous operation with every resisting structure in the depths of the sole; and, secondly, that he must be prepared to sever, in his open wound, every structure which he finds offering obstruction to the perfect and easy correction of the deformity.

An important element in the open method, and one to which Phelps strongly refers, is the need of "over-correcting" the deformity, if I may once more be allowed that expression. In the old operation the surgeon was content to lay down his tenotomy knife when, by the employment of a fair amount of force, he could bring the foot into a good position. He was content to leave a certain portion of the deformity to be effaced by the subsequent employment of Scarpa's shoe, or of some other retentive apparatus. But now, after operating according to Phelps's method, he fixes the foot in a gypsum casing, a good deal in the position of valgus, if not of calcaneo-valgus. As

a sailor might say, he gets "well to wind'ard" of the original defect.

In the paper which he read before the Berlin meeting of the International Congress, Phelps wrote that he had never found it necessary to resort to the open incision in children under one year of age. Had he postulated that the operation is not suited for infants, I would, in that one particular only, have joined issue with him. In suggesting the field of usefulness of an operation the age of a patient is without value as a definite landmark. Age is a purely relative term, and I fail to see why an operation which is good for a boy of eight years is unsuitable for one of eight months. Indeed, I have operated on an infant of eight months after this radical method, and with the most satisfactory results. This particular child was born with talipes equino-varus, in the private practice of a former house surgeon at the Children's Hospital, Dr. Bays, who had corrected much of the deformity by Achilles-tenotomy. He was familiar with the open method of treatment, and he called me in simply that the remaining deformity of this sturdy infant might be effectually dealt with once and for all.

Granting that the principle of the operation is good, I fail to see how the tender age of a child with congenital club-foot can of itself be a barrier against its adoption. What the surgeon desires is to correct the deformity at the earliest possible moment, with the least possible distress to the child, and with the greatest possible chance of shutting out the risk of relapse. For my own part, I have no "age limit," but I trust that it is superfluous for me to insist that I would not advise the open operation in a case which gave a fair prospect of yielding readily to simple subcutaneous tenotomy, with adjustments and massage; especially (as more frequently happens in private practice) when the child can be kept under the prolonged supervision of the operating surgeon, and under the well-directed manipulations of intelligent nurses or parents.

In one very severe case of relapsed equino-varus, after dividing all those fibrous structures which chiefly opposed the desired position of the foot, I deemed it expedient to complete the treatment by the open method by removing the astragalus. But the necessity of resorting to a tarsectomy in conjunction with the open operation, at any rate in the case of children, must be very rare. In a certain number of his cases Phelps has completed the open operation by dividing the neck of the astragalus, and in a smaller proportion he has gone a step farther, and has removed a wedge from the outer side of the os calcis. Of these modifications I have at present no personal experience, but I do not think that section of the neck of the astragalus can give much advantage over the insertion of a thick wedge of space into the astragalo-scaphoid joint. And if one is too ready to excise a piece of the os calcis there will be a risk of obtaining an improved position of the foot by the shortening of its outer border, rather than by the lengthening of the inner border. Still, I would not for a moment suggest that the open operation is never to be supplemented by a tarsectomy. Indeed, I think otherwise. But in the case of children, to which class of patients these practical observations are confined, the simple but thoroughly free incision on the inner side of the foot will usually be found to be sufficient. And if the results of treatment by lengthening the inner border of the foot can eventually be shown to be equal to those following the removal of a wedge of bone from the outer border, preference may be claimed for the simpler method.

Kirmisson records a case in the 'Journal de Médecine' (January, 1890), which he successfully treated by Phelps's operation, in which relapse had occurred after the performance of Lund's operation of removal of the astragalus. This I can well understand, for even after the removal of this bone the inversion of the foot in an extreme case is by no means effectually dealt with, unless at the same time the front of the deltoid ligament and the adjacent

tendons are also severed. And I am entirely in agreement with Kirmisson when he says that the thorough and open method of Phelps will probably render the removal of the astragalus, and tarsectomies in general, very exceptional procedures in the treatment of club-foot. Indeed, Phelps himself at the Berlin Congress insisted that osteotomy should never be resorted to as a primary operation. On the same occasion he also remarked that in none of the cases in which he had operated had a sensitive scar, a flat foot, or a local paralysis detracted from the usefulness of his treatment. And in a private note Dr. Phelps recently informed me that he had then performed the operation 200 times without a death.

So far as my own experience with the radical or open treatment goes (and I have practised it in a considerable number of severe cases of equino-varus during the last few years), I have every reason to speak well of it; and it is with the hope of bringing it into more general notice, as well as of learning the views which other surgeons hold concerning it, that I have ventured to bring the subject before this Society.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 26.)

ON SOME POINTS

IN THE

SUPRA-VAGINAL AMPUTATION OF THE CERVIX UTERI FOR CANCER,

WITH

SPECIAL REFERENCE TO THE METHODS ADOPTED IN CASES WHERE FOR TWO YEARS AND UPWARDS THE DISEASE HAS NOT RECURRED.

BY

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Received November 8th—Read December 13th, 1892.

The supra-vaginal amputation of the cervix uteri for cancer is an operation that has only been introduced into this country comparatively recently. Probably it is within the mark to say that it has only been practised here during the last ten years.

Briefly, the operation consists of removing the whole cervix at the level of the internal os, the bladder having been previously separated from the anterior aspect of the cervix to the level of the vesico-uterine pouch of peritoneum. A portion of the peritoneum forming Douglas's pouch, and corresponding to the posterior

aspect of the cervix, may or may not have to be removed at the same time.

I propose to consider the subject shortly under the following heads:

1. The indications and contra-indications for the operation.
2. Its mortality.
3. The results as regards recurrence.
4. The details of the operation in those of my cases where the disease has not recurred.

It should be stated that this paper is not intended as a comprehensive survey of the operation generally, but merely deals with my own experience of it, which consists of nineteen cases.

1. *The indications for the operation.*—The disease tends to spread—

(*a*) To the vagina;

(*b*) To the connective tissue round the cervix, thus involving the bladder, the broad ligaments, and the utero-sacral ligaments; and

(*c*) To the body of the uterus.

So far as my own observation of cases and specimens goes, the tendency to spread in this last direction is comparatively slight. Even when the disease has spread to the body of the uterus, it has in most cases, though certainly not in all, already spread so extensively either to the vagina, or into the connective tissue round the cervix, that complete removal of the diseased tissue has become impossible, and not to be accomplished even by removing the whole uterus.

The object of any operation in these cases is of course to give the patient immunity from the disease, either completely, or at any rate for a considerable period; and unless the intended operation, whether it be the supra-vaginal amputation of the cervix or total extirpation of the uterus, can so thoroughly remove all the parts affected as to give a good chance of this, it does not seem to be worth doing.

For the success of the supra-vaginal amputation it is generally essential that the growth should not have spread to the vaginal walls, nor have involved the connective tissue round the cervix. A very limited extension of a *superficial* growth on to the vagina is not always a contra-indication, as it may be met now and then by making the incisions sufficiently widely, and afterwards applying the cautery thoroughly to the cut edge of the vaginal wall towards which the growth was adjacent. Even a limited extension of the growth to the vagina, while not an absolute contra-indication for the supra-vaginal amputation, is, however, of highly unfavorable augury as regards recurrence.

Extension of the disease into the connective tissue round the cervix is usually an absolute contra-indication when it can be clearly recognised to have occurred. This often cannot be done with certainty. I refer to that group of cases where the uterus is very fairly movable and where there are the so-called "thickenings," or bands of a more or less definite character, to be felt running outwards from the cervix, either laterally in the direction of the broad ligaments or backwards in the direction of the utero-sacral ligaments.

Bands felt in the situations mentioned may be due to extension of the disease; but bands of this unfavorable import may be, in certain cases, closely simulated (*a*) by the cicatricial bands or bridles so commonly met with after the menopause as to be within the limits of normal; (*b*) by the thickening resulting from old inflammatory mischief, parametritic and perimetritic; and (*c*) in the case of bands in the situation of the utero-sacral ligaments by an unusually definite condition of these ligaments, apart from inflammation, or as the result of it. Every gynæcologist must be familiar with cases where, apart altogether from malignant disease of the uterus, the utero-sacral ligaments have been unusually well defined.

In the group of cases referred to, therefore, where there are such bands, and where the uterus is very fairly

movable, I think it best to give the patient the benefit of the fairly even chance (that the bands may not indicate extension), and perform the operation.

Every now and again a case may be met with where a very limited extension of the disease into one broad ligament may be successfully dealt with by extirpating the uterus and placing a pair of large pressure-forceps for forty-eight hours on the broad ligament in question, as far outwards as appears safe, with the object of causing sloughing and so completely removing the disease. I have had a case of this kind, and up to the present time—two years since the operation—no recurrence has taken place. I hope to record this case fully on another occasion.

Both the supra-vaginal amputation of the cervix and total extirpation of the uterus are absolutely contra-indicated when, for any reason whatever, the uterus cannot be drawn down so that the os uteri is on a level with the orifice of the vulva.

As regards the cases where extension has taken place towards the uterus, the vagina and connective tissue round the cervix not being involved, vaginal extirpation of the uterus is indicated, and supra-vaginal amputation of the cervix contra-indicated. The fact of this extension cannot usually be recognised except at the time of operation, and therefore in every case one should begin the operation prepared to do either the supra-vaginal amputation or total extirpation.

The mortality of the supra-vaginal amputation.—The mortality has a special interest as bearing on the question whether the supra-vaginal amputation of the cervix or total extirpation of the uterus should be the usual operation for cancer of the cervix. As has been said above, it is not always possible to tell whether or not the body of the uterus has been invaded by the growth. This being so, it is argued that if high amputation of the cervix and vaginal hysterectomy have an approximately equal mortality, it is better to remove the whole uterus on the chance that the body of it may be involved. While

recognising that the mortality of total extirpation has been considerably reduced of late years, it still seems to me that removal of the whole uterus is a more serious operation than the supra-vaginal amputation of the cervix, and the results of my own cases bear out this opinion. My results are as follows :—I have performed the supra-vaginal amputation of the cervix nineteen times without a death, and removed the whole uterus for cancer *per vaginam* six times with one death. Three of the total hysterectomies were for primary cancer of the body of the uterus, and all these recovered; three were for cancer of the cervix, and one of these died.

As regards recurrence.—My first ten cases of supra-vaginal amputation of the cervix for cancer were recorded in the 'Lancet' of March 10th, 1888. Three of them were in good health and free from recurrence for two years and upwards after the operation. One of these three (No. 9 in the table) had recurrence about three years after, and died. One of them (No. 3) has not been seen since two years after the operation, when she was in good health and without recurrence. One of them (No. 6) was in good health and free from recurrence when I last saw her three years after the operation. Quite recently (October 3rd, 1892) Dr. Dundas Grant (who sent this case to me in the first instance) told me that he had lately seen her, and that she was still quite well, though it is now more than five years since the operation.

As regards the remaining nine cases in which I have performed the supra-vaginal amputation, the results are as follows:

In one (No. 19) the operation was incomplete, *i. e.* I felt certain that the disease had extended beyond the limits of the part removed.

In the remaining eight cases the operation was complete. In four of these (Nos. 12, 16, 17, 18) the patients are well and free from recurrence.

No. 12 was operated upon on May 31st, 1888—an example of the cauliflower growth; microscopical sec-

tions show it to be a typical squamous epithelioma. I last examined her on October 7th, 1892, and found her free from recurrence. I may add that the sections have been examined by various independent observers; among others by Dr. John Williams and Mr. Butlin.

This patient was sent to me by Dr. Cursham Corner, of Mile End, who was present at the operation.

No. 16, who was three months pregnant at the time of the operation, is still quite well and free from recurrence. The operation was performed at the London Hospital on August 26th, 1889. I have seen this patient regularly at least once a month up to the present time, and examined her from time to time. There has never been any sign of recurrence.

A drawing of the specimen and of a section of it accompany this paper. The growth is a typical example of a columnar epithelioma. Sections of this also have been examined by many independent observers, among them by Dr. John Williams and Mr. Butlin.

No. 17 was a private case. I have not examined her since the operation, which was done on February 18th, 1890; but I have seen a letter from her, dated two years after the operation, in which she says she is in good health and free from symptoms. It so happens that quite recently I found that one of the sisters at the London Hospital knows the patient very well, and often sees her. She confirms the patient's account of herself.

A drawing of the specimen, and of a section of it, accompany this paper. The growth is an example of columnar epithelioma.

In these three cases, then, the evidence, on the one hand of malignancy, and on the other of the non-recurrence of the disease, is quite conclusive.

As regards No. 18, it is a case where there may perhaps be two opinions as to the nature of the growth. Sections of it under the microscope show numerous glandular cavities, lined by a single layer of cylindrical epithelium. My own belief is that clinically it was

malignant. The symptoms and physical signs were in favour of its being so; but still more convincing to my mind was the fact that extension had taken place on to the anterior vaginal wall. The portion of the anterior vaginal wall thus involved was removed at the time of operation, and sections of it show the same glandular cavities as sections from the growth in the cervix itself. As no such glandular cavities are normally present in the vaginal wall, this extension seems very hard to explain, unless the growth itself was of a malignant nature.

The operation was done on May 7th, 1891, and at present the patient is quite well, and there is no sign of recurrence.

To return to Cases 12, 16, and 17, where the evidence of malignancy is conclusive, and where no recurrence has as yet taken place, the details are as follows:

CASE 12.—Mary C—, æt. 42, at that time matron of the East London Nursing Association, was sent to me on the 19th of May, 1888.

She had been married nineteen years, and had had two children, the last sixteen years ago. She had had no miscarriages. She had pelvic peritonitis after the second confinement.

History of the present illness.—For the last two or three months she has had pain in the lower abdomen, especially in the left iliac region on walking. The pain shoots down the left leg. She is always easier on lying down.

Five weeks ago she had a discharge from the vagina "like coffee grounds." It was offensive. This discharge continued till a week later, when the ordinary menstrual period came on.

Though it is now a week since her last period, she is still losing a brown discharge.

She did not think she had been getting thinner.

The catamenia have usually been regular every four weeks, lasting six days, and attended with a good deal of pain in the left iliac region for three days before and

during the first twenty-four hours of the period, but not afterwards.

For many years she has been subject to a white discharge between the periods, but for the last two or three months the intermenstrual discharge has been yellow, or sometimes brown.

On examination a flat "cauliflower" growth was found on the posterior lip of the cervix. It was raised about one sixteenth of an inch above the adjacent healthy surface, and occupied an area roughly about the size of a two-shilling piece. It bled readily on touching it. The uterus was freely movable.

She readily consented to have done whatever might be thought desirable, and accordingly, on May 31st, 1888, I performed the supra-vaginal amputation of the cervix at the London Hospital.

The chief points requiring mention as regards the operation are that the incisions made through the vaginal walls, in order to free the cervix, were made as far from the growth as seemed safe; also that it was found desirable to incise the perinæum, so as to afford ample room for the various manipulations. Douglas's pouch was not opened. No ligatures were used, but pressure-forceps were left on bleeding vessels. These were removed in twenty-four hours.

The length of the cervix measured, after being six days in spirit, 2¼ inches.

On June 7th, in order still further to increase her chance of immunity from recurrence, I applied Paquelin's cautery to the posterior and lateral aspects of the wound left after removal of the cervix.

The patient's recovery was quite uneventful: the temperature only once reached 100°, which was on the evening of June 2nd; on other occasions it was never above 99·5°.

Before the operation her weight was 8 st. 11½ lbs.; and when I last saw her, October 7th, 1892, it was 9 st. 10 lbs. I examined her on that date, and found nothing to indicate recurrence.

DESCRIPTION OF PLATE I.

On some Points in the Supra-vaginal Amputation of the Cervix Uteri for Cancer (Dr. ARTHUR H. N. LEWERS).

FIG. 1.—A drawing of the cervix removed in Case 12 (Mary Core, The Hollies, Blandford Road, Ealing). The view is that that would have been obtained through the speculum before removal of the cervix. A "cauliflower" growth is seen growing from the posterior lip. Date of supra-vaginal amputation of the cervix, May 31st, 1888.

FIG. 2.—The cervix removed by supra-vaginal amputation in Case 16 (Mrs. Austin). Side view of the specimen. A small glass rod has been passed along the cervical canal. Date of operation August 26th, 1889.

FIG. 3.—The cervix removed by supra-vaginal amputation in Case 17 (Mrs. Burne, Wasing Rectory, Reading). The view is that which would have been obtained through a speculum before the operation. A soft "cauliflower" growth is seen attached chiefly to the right side of the vaginal portion. Date of operation February 18th, 1890.

For the microscopic appearances of the growth in these figures see following Plates.

Fig. 1.

Fig. 2.

Fig. 3.

DESCRIPTION OF PLATE III.

On some Points in the Supra-vaginal Amputation of the Cervix Uteri for Cancer (Dr. ARTHUR H. N. LEWERS).

FIG. 1.—A portion of the growth shown in Plate I, fig. 2, under a high power. (Hartnack, obj. 8, eye-piece 2.)
It is a typical columnar-celled epithelioma. From Case 16.

FIG. 2.—A portion of the growth shown in Plate I, fig. 3, seen under a high power. (Hartnack, obj. 8, eye-piece 2.)
An example of columnar-celled epithelioma. From Case 17.

Fig 1

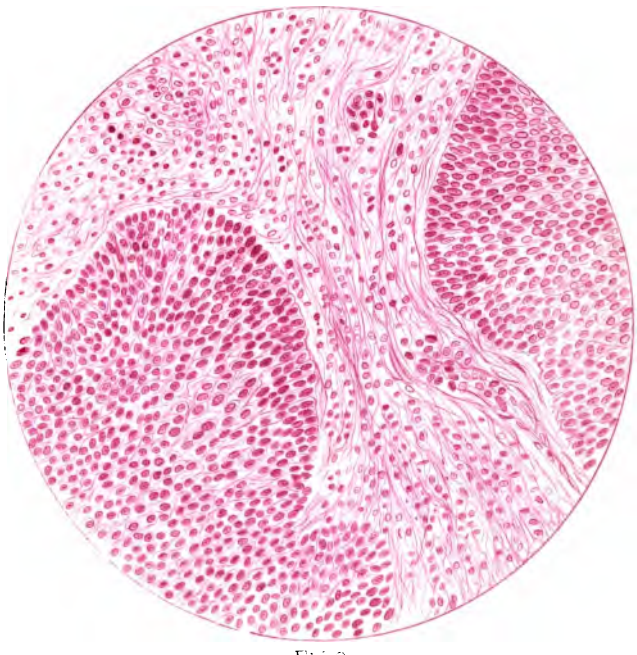

Fig 2

There is in this case unfortunately some tendency to alcoholism, and she has been from time to time ill with gastric catarrh, due, I believe, to this cause, for a week or so at a time; otherwise she has been quite well and able to do her work ever since the operation.

CASE 16.—Eliza A—, æt. 40, was admitted into the London Hospital on the 21st of August, 1889.

She had been married twenty-one years, and had had eight children and two miscarriages. The last confinement was a year and nine months before admission, and the last miscarriage four years ago.

History of the illness.—In October, 1888, she began to suffer from a discharge, at first white, but afterwards yellow, and with a little blood in it constantly. In March, 1889, she had an attack of severe flooding, and she has had similar attacks every month up to the time of her admission. Large clots were passed each time.

The catamenia appeared when she was ten years old; she then "saw nothing" till she was between thirteen and fourteen, when she became quite regular every four weeks.

Her appetite has been bad since the beginning of the illness.

State on admission.—The external genitals and neighbouring skin are inflamed simply, as they become in patients who are the subjects of a constant discharge.

The vaginal portion of the cervix is considerably enlarged in circumference. There is an irregular nodular growth, chiefly involving the posterior lip. It is hard but brittle, and bleeds readily on touching it. The growth has a yellowish colour. The uterus was freely movable and somewhat enlarged, though its exact size was not made out well till just before the operation, when the patient was under the influence of an anæsthetic. The patient was very fat, and her weight was 12 st. 3 lbs.

On August 26th (1889) the supra-vaginal amputation

of the cervix was performed. When the cervix had been freed up to the level of the internal os, it was cut off at that level with Paquelin's cautery ; and the cautery was also applied to the posterior and lateral aspects of the wound. In freeing the posterior aspect of the cervix Douglas's pouch was opened to a small extent, the opening being about a quarter of an inch in length transversely.

The length of the cervix after removal was one inch and a quarter.

Four pairs of small pressure-forceps were left on bleeding vessels. They were taken off twenty-four hours later.

The highest temperature was 100°, which was reached on four occasions—the evening of the 2nd, 3rd, 5th, and 7th days. The pulse rate never exceeded 85.

It may be mentioned that the examination just before the operation showed that she was about three months pregnant, and she miscarried on the evening of August 29th.

The patient did quite well, and left the hospital on September 12th.

The fact that she was pregnant at the time of operation caused the parts to be unusually vascular ; and besides, in general, pregnancy is an unfavorable complication as regards recurrence, the physiological activity of the pregnant condition appearing to favour an early infiltration of the surrounding tissues.

However, in this particular case the patient, who has been very regular in coming to report herself at the hospital, is still quite well, now over three years since the operation, and there is no sign whatever of recurrence.

Case 17.—Mrs. B—, a clergyman's wife, 53 years of age, was first seen by me on the 24th of January, 1890. She had had two confinements, the last twenty-two years ago.

She was regular till about three years ago, when the menopause occurred. She has been feeling weak ever since

last summer (1889), and since that time has had a yellow vaginal discharge, which has been blood-stained from time to time. She has had no pain.

On examination, a cauliflower-like growth, about the size of half a walnut, was found attached to the right half of the cervix. It was very brittle, and bled readily on touching it.

A few days later the patient was examined carefully under an anæsthetic with the object of ascertaining the fitness of the case for operation. The uterus was quite freely movable, and there was no evidence of the disease having spread beyond the limits of the cervix.

The patient was advised to have either supra-vaginal amputation of the cervix performed, or vaginal hysterectomy, according to what might seem necessary at the time of the operation.

She readily consented, and accordingly the operation was done on the 18th of February, 1890.

The points in the operation that require particular mention are, that Douglas's pouch was very freely opened, the opening being an inch in measurement transversely. The cervix was removed at the level of the internal os, and it was immediately slit up to see how near the obviously diseased part was to the internal os. There seemed to be an ample width of apparently healthy tissue between the points mentioned, and accordingly I decided to be content with removing the cervix, although it would have been very easy to have removed the remainder of the uterus, the body of it being quite small from senile atrophy.

Although Douglas's pouch had been opened so freely, I applied Paquelin's cautery to the right aspect of the wound. It should also be mentioned that the cervix was cut off from the body of the uterus by the cautery, after having been freed from its attachments up to the level of the internal os in the usual way.

The opening in Douglas's pouch was closed with one suture, and a small india-rubber drainage tube was left,

so that one end was just within the peritoneal cavity, and the other in the vagina. The tube was found loose in the vagina on the next day.

Pressure-forceps were left on bleeding points for thirty-six hours. The patient subsequently did very well, the highest temperature was 101° (on the evening of the second day), and after that it did not rise above normal.

Among others present at the operation was Dr. Hugh Smith, of Englefield House, Highgate, who was at that time Resident Accoucheur at the London Hospital.

As above mentioned I have not seen this case since the operation, but I have seen a letter from her dated February 17th, 1892, saying she was then (two years after the operation) quite well and free from symptoms.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 34.)

THE EFFECTS OF THE IODIDES

ON

ARTERIAL TENSION AND THE EXCRETION OF URATES.

BY

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In vol. lxxi of the 'Transactions' of the Society there is a paper of mine on the effects of certain drugs which cause retention of uric acid, in which special mention is made of salts of iron, lead, and lithium.

Since that time (1888) the list of substances having a similar action on the excretion of urates has been, as we shall see presently, greatly extended, and I desire to show in the following paper that yet another group must be added to the list, namely, that containing certain well-known iodides, hydriodic acid, and probably iodine in other forms.

I believe that the action of these compounds on the excretion of urates is of great interest, and will eventually be found to account for a very large part of their well-known action in medicine and surgery, and it may be remembered that I have already made a similar remark with regard to two other very important drugs, mercury and opium, which act, as we shall see presently, in exactly the same way that iodides do, but I shall content myself

in this paper with pointing out the facts as they have come to my knowledge, and with mentioning shortly a few of the more important points in which they may help us to understand more completely the action of these substances in disease.

My first experiments with iodides led me to believe that they had but little action on the excretion of urates, and I say in my Thesis on uric acid, published in the 'British Medical Journal,' 1888, vol. ii, p. 11, "Iodide of Potassium has, in five-grain doses, but little action on the excretion of uric acid; in larger doses it acts apparently merely as an alkali. Iodine taken as the tincture has little or no direct effect."

But when, later on in my researches, I came upon the relation of the excretion of water to the excretion of urates, which in "uric acid"[1] p. 100, I have stated as follows:—"the urinary water varies inversely as the uric acid excreted along with it," a relation which I believe holds both in health and disease, and throughout the action of a whole series of drugs (see 'Journal of Physiology,' vol. xiii, p. 300), it at once struck me as a very extraordinary thing that the iodides of potassium or sodium which have such a marked and immediate effect on the excretion of water should have no effect on the excretion of urates.

In fact, that they should resemble in their action on the excretion of water a large series of drugs which clear the blood of uric acid and diminish its excretion in the urine, and yet differ from them in having no action whatever on the excretion of urates.

I therefore began to re-investigate the matter, and I soon found that there was no exception to the rule, and that when iodides produced a diuresis they produced also, like all the other drugs in the group, a diminished excretion of urates; and I had previously overlooked this fact because, not knowing the relation of water to urates in

[1] 'Uric Acid as a Factor in the Causation of Disease,' J. and A. Churchill, 1892.

excretion, I had not observed the diminished excretion of urates under iodides, or had attributed it to something else. And there was one other fact which I am now quite familiar with, but did not, perhaps, sufficiently bear in mind during my first experiments with iodides, namely, the relation of the excretion of urates to the acidity of the urine.

In 'Uric Acid,' p. 97, and in previous writings, I have stated the rule thus:—"The excretion of uric acid varies inversely as the acidity; its greatest hourly excretion occurs in the alkaline tide of the morning, and the smallest hourly excretion in the acid tide of the night—facts which are very well known to physiologists."

Now this law is practically an absolute one, and holds in everyone in all physiological and in most pathological conditions also, and by altering the acidity, it is not only possible to alter the excretion of urates from hour to hour and from day to day in anyone;[1] but by completely altering the cycle of acidity, we can completely prevent both the plus excretion of urates in the alkaline tide, and the minus excretion in the acid tide of the night; they are simple results of the normal cycle of alterations in acidity.

Now, in my early experiments with iodides, I overlooked the fact that on several occasions the acidity fell without the uric acid rising, as it should have done in accordance with the above law: and that on other occasions, when the iodide was left off, the uric acid rose in spite of the acidity remaining the same as on the previous day.

These results should have opened my eyes to the effects of iodides on the excretion of urates; but I did not then fully realise the importance and wide application of the rules I have mentioned.

Another exception to the rule of acidity is seen in the case of the salts of lithium (see 'Transactions,' vol. lxxi, p. 287), but the explanation of this is simple enough (see also 'Uric Acid,' p. 30). And mercury, lead, iron, and

[1] See also my experiments on dogs, 'Uric Acid,' p. 53.

all other metals which form insoluble compounds with uric acid, diminish its excretion and produce a diuresis without any regard or even in opposition to acidity, and the iodides act like one of these, because directly or indirectly they influence in the same way the solubility of uric acid in the blood.

It follows from the above-mentioned rules that arterial tension varies with the amount of uric acid that is being excreted in the urine, and this varies with, and is an index of, the amount of urate circulating in the blood.

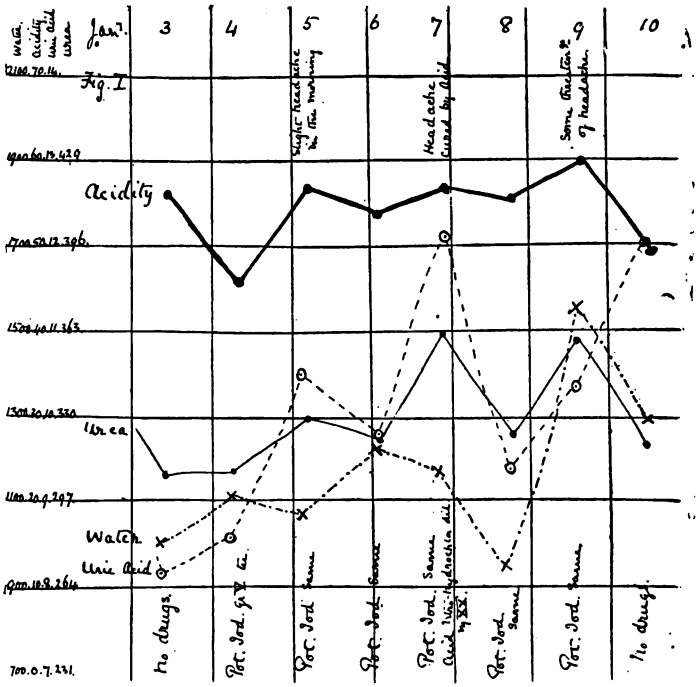
But arterial tension means contracted arterioles, and contracted arterioles mean that water has difficulty in passing the kidneys, as is shown to be the case in the parallel action of digitalis and other drugs which contract the arterioles (see 'Uric Acid,' p. 100) and this is the reason why the urinary water varies inversely as the uric acid in the urine, because contraction of arterioles varies directly with the uric acid in the blood.

My results bearing on the effects of iodides on the excretion of urates, were for a time also rendered of doubtful value by the fact that when an iodide is present in a specimen of urine, and a solution of nitrate of silver is added to that urine, as in Haycraft's process for the "Estimation of Uric Acid" ('British Medical Journal,' 1885, vol. ii, p. 1100), which is the one I use, the silver combines first with the iodide and then with the urate, so that if care is not taken to add enough silver to combine with all the iodide as well as all the urate, the quantity of urate present may be greatly under-estimated.

But, acting on the kind advice of Mr. J. E. Saul, F.I.C., to whom I have often been indebted for help on points of chemistry (see 'Uric Acid,' pp. 30 and 36), I found that by adding the silver solution till no more iodide came down, and then further adding the quantity usually required for the process, the whole of the urate could be obtained with absolute certainty, so that my results are not now complicated by errors arising from this action of iodides on the process of estimation.

In Fig. 1, I show the effect of five grains of iodide of potassium taken three times a day for six days.

FIG. 1.



The effect on the excretion of uric acid is certainly not very marked, but, with the exception of January 7th, the uric acid curve is below or close to the urea curve the whole time the drug is being taken.

On January 4th the uric acid rose a little, but not as much as one would have expected from the fall of acidity, and on January 10th, after the drug had been left off, it rose very decidedly, though this corresponded with a fall of acidity; but the difference between the slight rise on the 4th and the great rise on the 10th, with a fall of acidity in both cases, is very marked.

On three days, the 5th, the 7th, and the 9th, there

was more or less headache, and on the 7th, with the largest excretion of urate, it was so bad as to interfere with work, and some acid had to be taken to cure it.

Now, some headache is in my case a very common sequence to a few doses of an iodide, and I believe it is really of the nature of a rebound, that is to say, that just as occurs with mercury and other metals, or with opium, the minus excretion of urate which is their first effect is due to a storing up of urate in the body, and the natural result of this is, that when the urate is again able to pass into the blood, it is present in some excess in that fluid, and gives rise to symptoms (constituting what I have spoken of as the rebound), which are for the most part the reverse of those present during the first action of the drug.

Thus, during the first action of these drugs there is a diminished excretion of urate, the pulse is quick and soft, the mind is clear and tranquil, and the urine is more or less profuse; during the secondary action or rebound there is a plus excretion of urate, the pulse is slower and harder, there is mental depression and more or less headache, and the urine is scanty.

It will be noticed that there was a threatening of headache on January 9th, though uric acid was below urea, and it may be thought from what I have said, that there would be no excess of uric acid in the blood during any part of that day; but the point in each curve represents the excretion of an entire twenty-four hours, and the excretion of urate followed from hour to hour (see 'Uric Acid,' fig. 1, on p. 16) shows endless fluctuations, in fact it may truly be said, that it is never the same for two hours together; and the point in the curve represents merely the balance of these fluctuations during twenty-four hours.

If the relation of uric acid to urea in the twenty-four hours' excretion is exactly 1 to 33, the two points are superimposed, as is almost the case on January 6th. On January 5th the upward fluctuations of urate excretion exceeded the downward by about half a grain, and on January

7th by about one grain; on the 9th, on the other hand, the downward fluctuations exceeded the upward by about half a grain; but this does not show that uric acid was above urea the whole day on the 5th and 7th, or below it the whole day on the 9th; and, followed from hour to hour, the headache and other symptoms mentioned will be found to correspond absolutely with the excretion of urate, so that it is by no means extraordinary that there should be a slight headache for some hours on the 9th, but the headache was more marked and decided with the large excretions of the 5th and 7th, just as we should expect.

I may explain that, for reasons given in 'Uric Acid,' pp. 9—13, the height of the uric acid above the relation to urea of 1—33 is the index of the amount of uric acid passing through the blood on any given day; *i. e.* on January 5th half a grain passed through, on January 7th about a grain. On the day of a uric acid headache, as much as 5 or 6 grains may pass through, *i. e.* the uric acid exceeds the relation to urea of 1—33 by 5 or 6 grains, and the effects on the arterioles and the excretion of water are proportionate to the amount of urate thus passing through the blood (see 'Transactions' of the Society, vol. lxx).

Precisely the same remarks apply to the curve showing the excretion of water. The crosses represent again the balance of the twenty-four hours' fluctuations; and, taken from hour to hour, the excretion of water keeps up in the most remarkable manner its inverse relation to the excretion of urates; but, regarding the excretion of water, one point in which it differs from the other curves must be borne in mind.

The effect of clearing urates out of the blood is to relax the arterioles all over the body, those of the kidney among others, and the effect of relaxing the arterioles of the kidney is to allow any excess of water in the blood and tissue fluids to run off; but supposing there has been already a drain of water from the body in perspiration, diarrhœa, vomiting, or a diuresis, then clearing the urates

out of the blood and relaxing the arterioles will produce little or no effect on the excretion of water.

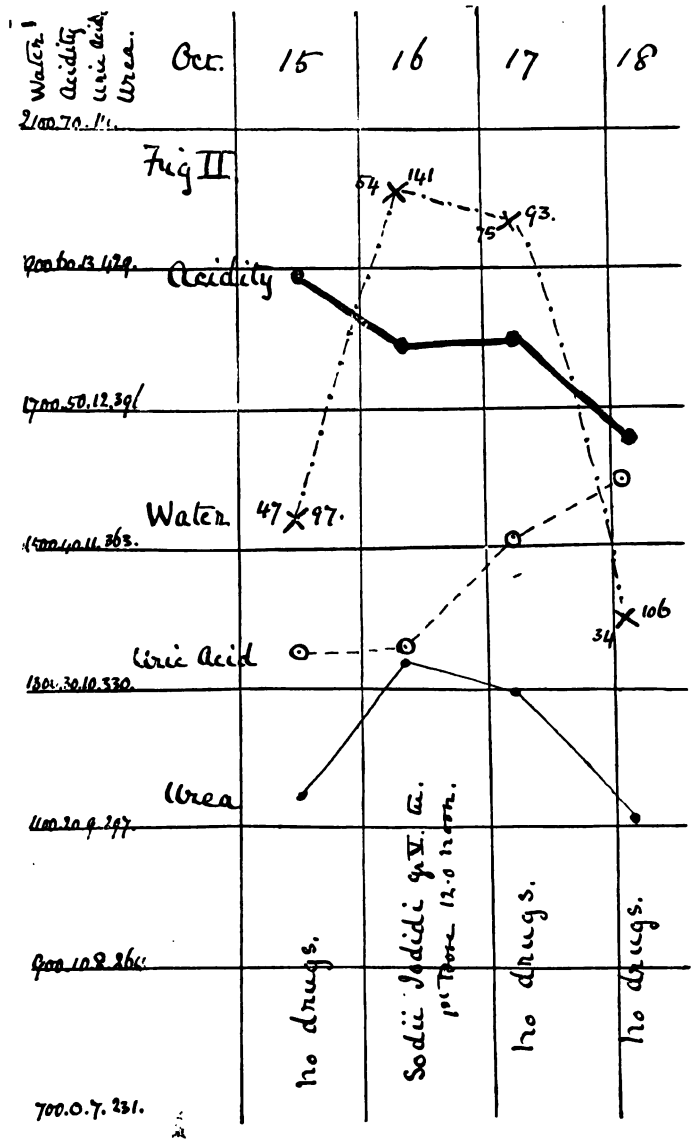
And the converse of this also holds; and when from chronic excess of uric acid in the blood with contraction of the arterioles, as in Bright's disease, water has been gradually for days and days accumulating in the blood and tissue fluids, constituting what we call dropsy, if the blood is cleared of urates and the arterioles are thus allowed to relax, we get a correspondingly large and continuous excretion of water till the dropsy is all gone. For further argument on this interesting subject, see my paper on "The Elimination of Water and the Pathology of Dropsy," 'Lancet,' July, 1892.

And the point on which I desire to lay special stress with regard to the excretion of water is, that the moment the arterioles are relaxed, say perhaps only for two hours in one day, the rush of water in those hours may be so great as to produce a considerable effect on the excretion of the whole twenty-four hours; while the corresponding fluctuation in urate excretion may seem insignificant by comparison. But the point about the urate excretion is that for two hours the blood was practically cleared of urate and the arterioles were able to relax; and the point about the excretion of water is that at the time the arterioles were able to relax, there was a large amount of water in the blood and tissue fluids, which could be run off. It is necessary to bear these points in mind, or very incorrect conclusions may be drawn from the facts.

In Fig. 1 the water rises on the 4th with the low excretion of urate, it falls a little on the 5th when urate gets above urea, rises again on the 6th as the urate falls, falls on the 7th and 8th in connection with the large excretion of urate on the 7th, and rises again sharply on the 9th with the low urate excretion on the 8th and 9th, to fall again on the 10th as the urate goes up, thus illustrating very well what I have said, that the urinary water is from day to day and hour to hour inversely as the urate excreted along with it.

Fig. 2 shows the effect of five grains of iodide of sodium taken three times on one single day.

FIG. 2.



I should say that where my curves relate to twenty-four hours the days are divided from each other at 7.0 a.m., that is to say, the excretion of the 15th October is the excretion of the twenty-four hours ending 7.0 a.m. on the 15th.

It will be noticed that the curve representing the excretion of water in this and the remaining figures has numbers at each side of the crosses; the number at the left hand side of the crosses, 47 on October 15th, represents the hourly excretion of water in c.c. during sixteen hours of the day ending 11.0 p.m., and is obtained by dividing the total urine passed in this period by 16. The number on the right hand side of the cross, 97 on October 15th, represents the hourly excretion in c.c. during the eight hours ending 7.0 a.m, and is obtained by dividing the total excretion of those hours by 8. As a rule the hourly excretion is less in the sixteen hours of the day than in the eight hours of the night, because the urate excretion is just the reverse of this.

But, as I have said, the urate excretion is dependent on the acidity, and by altering the acidity we can alter the excretion of urate so that it is no longer most in the day and least in the night, and when we do this we shall find that we have also altered the excretion of water so that it is most in the day and least in the night, the reverse of the normal condition. (See 'Brain,' spring and summer number, 1893, p. 235.)

In Fig. 2 we see that on the day the iodide was taken there was a rise in urea, a fall in acidity, and stationary uric acid. (In Fig. 1 also the urea tended to rise all the way from January 4th to 9th.)

We should have expected uric acid to rise, because acidity fell; but it did not. On the 17th, however, it rose decidedly, though acidity did not fall. On those two days, then, it was dissociated from its usual relation to acidity, and it was so on account of the iodide administered; and the same thing is seen on some of the days in Fig. 1.

In Fig. 2 the effect on the excretion of water is very marked, for on October 16th it rose sharply, being 54 c.c. per hour in the day and 141 c.c. per hour in the night, that is to say, the great diuresis took place at night; but the first dose of iodide was not given till 12.0 noon, when the large urate excretion of the alkaline tide and its correlated scanty excretion of water had been going on for some time; but for this the total urate excretion would have been less, and the water during the day hours more profuse.

The diuresis of the night reached such great proportions, not because the arterioles were greatly relaxed, but rather because, at the time they were relaxed the amount of water in the blood and tissue fluids available for excretion was large.

On the 17th the uric acid rose, but this was probably due to a plus excretion in the evening and night, as the water in the day hours was not scanty.

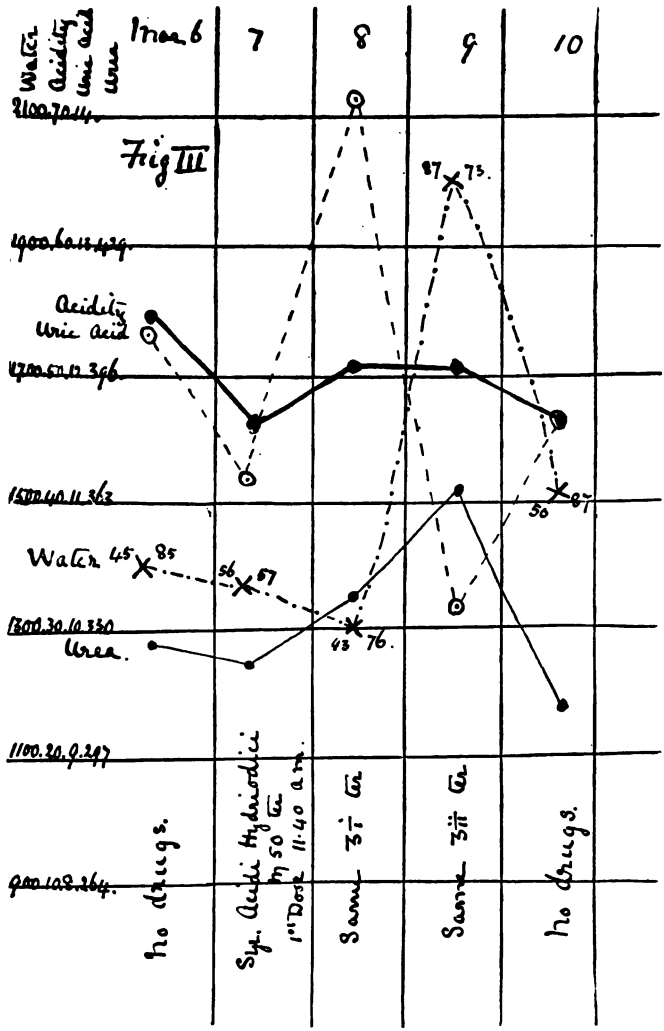
Later, on the 18th, the uric acid rose still further, and, as the effect of the iodide had now passed off, there was a plus excretion in the morning at the usual hours, and the urinary water was extremely scanty, in the day period only 34 c.c. per hour.

It is obvious then, that by thus observing the hourly excretion of water, and by applying the rule of its inverse relation to the excretion of urate, we can tell fairly well when a plus excretion of urate on any given day took place; as a rule, and apart from the action of drugs, a plus excretion of urate on any given day means a plus excretion in the alkaline tide of that day, and the water is correspondingly scanty at that time. But either drugs or disease may upset the normal cycle and cause a plus excretion of urate in the evening and night as well, and then the excretion of water is scanty in these hours also; and the contrast between the day excretion of water on the 17th, when the iodide was still diminishing the excretion of urate, and that on the 18th, when its effects had come to an end, is very marked, though, no doubt

also, the small excretion of the 18th is partly due to the fact that the blood and tissues were then rather poor in water, owing to the diuresis of the previous days.

Fig. 3 shows the effect of syrup of hydriodic acid taken

FIG. 3.



for three days, in doses increasing from ɱ50 on the 7th to ʒij on the 9th, each three times a day. With the smaller doses the effect was slight, and there was but little iodide in the urine; on the 9th, with a larger dose, there was a well-marked precipitate of iodide of silver, the excretion of urate was greatly reduced, and there was a correspondingly great diuresis.

If anyone were to take the position that the fall of urate was due to an error in the process, that is, due to the iodide in the urine, while the excretion of urate was really not affected at all, I should point to the large excretion of water as evidence that the excretion had really been diminished, and the arterioles relaxed.

Here, as in the other figures, the fall of urate on the 9th is out of all proportion to the rise of acidity on the previous day which is maintained, and the rise of urate on the 10th is equally out of all proportion to the trifling fall of acidity.

The water again is pretty constant in its inverse relation to the urate, the smallest water excretion of any day being on the morning of the 8th with the largest excretion of urate, and the largest excretion of water on the morning of the 9th, with the smallest excretion of urate; so that, as I have often remarked, those who have no means of estimating uric acid can really infer its fluctuations with great accuracy from the corresponding fluctuations in the excretion of water, if these are carefully measured.

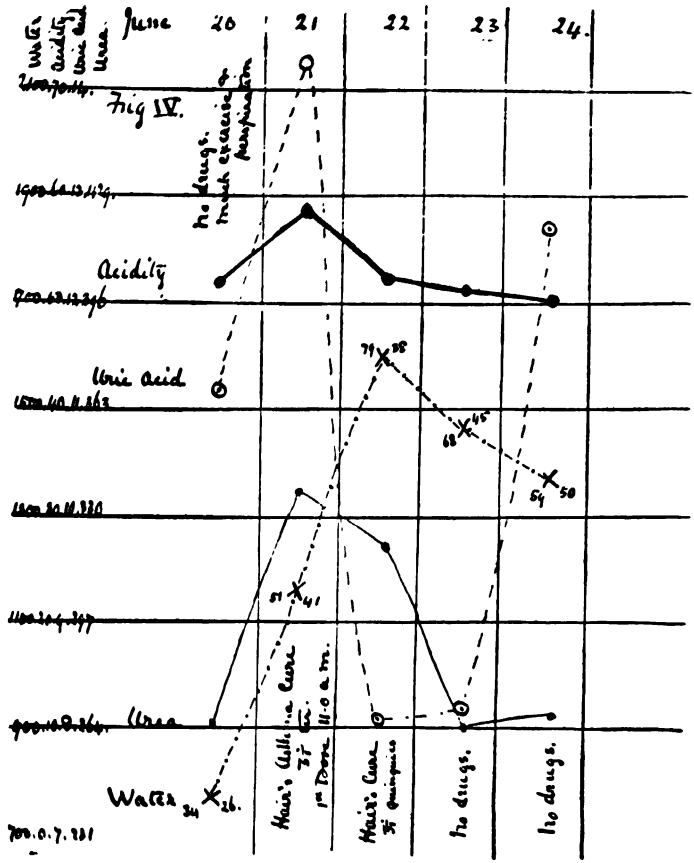
Fig. 4 shows the effect of doses of Hair's asthma cure, which is well known to contain an iodide, and it will be seen that it brought down the urate very low on the 22nd June, and kept it down on the 23rd with a correspondingly marked diuresis.

It is remarkable that there should be a large excretion of urate on the 21st; but this is not due to the iodide, but to the fact that it was overpowered by alkali.

The sequence of events should, I think, be read as follows:—On June 20th much exercise was taken, and

this caused a good deal of perspiration; on the 21st as a result of the exercise there was a very marked rise of urea, but the great loss of acid in perspiration prevented

FIG. 4.



the acidity from rising so high as it otherwise would have done, for urea and acidity generally rise and fall together, and as the acidity did not rise much, the excretion of urate was large in the alkaline tide of the 21st.

This shows very well the effect of exercise and the perspiration it produces in preventing gout, as noticed by

Cullen and others; for if the rise of urea on the 21st had been due to indulgence in nitrogenous food, the acidity would have risen to a corresponding extent, and some urate would have been held back and retained in the body; but with the rise of urea due to exercise there was a free excretion of urate; hence exercise tends to prevent gout by providing for a free excretion of the urate produced (see 'Uric Acid,' p. 149—236).

On the 22nd of June the iodide came into action, and the urate came down with a run in spite of falling acidity. On the 23rd it remains low because the iodide is still in the circulation, but on the 24th it runs up very high with scarcely any fall of acidity because the action of the iodide has come to an end.

With regard to the excretion of water, it is very low on the 20th because of the removal of much fluid in perspiration on that day.

It rises very markedly on the 22nd with the fall in uric acid, and here again the excretion of water bears witness to the accuracy of the urate estimation. It was profuse in the day hours of the 22nd, and scanty at night, probably because nearly all the available water had been excreted in the day hours; on the 23rd with a smaller excretion in the day, there was more at night.

I have on several occasions taken tincture of iodine, but it did not appear to influence either the excretion of urate or the excretion of water in the same way as the above-mentioned iodides.

In doses up to ηvii three times a day there was no iodide of silver formed on adding silver solution to the urine next day; the only effect of such a dose seemed to be the production of some gastro-intestinal irritation and dyspepsia with epigastric pain, nausea, and flatus; and if these were severe they produced their usual effects, namely, a fall in urea and acidity and a corresponding rise in the excretion of urate; therefore, tincture of iodine either has no effect at all, or indirectly increases the excretion of urate, and, like colchicum, which acts in

exactly the same way, it should be useful in acute gout: but for my part I greatly prefer to cure gout by other means, and to leave the gastro-intestinal mucous membrane intact (see 'Uric Acid,' p. 165).

It appears, then, that the iodides diminish the excretion of uric acid in the urine by causing it to be held back or retained in the body; and that this their first action is followed by a rebound with a plus excretion of uric acid.

Professor Latham says ('Lancet' vol. i, 1885) that iodide of potassium decomposes uric acid, splitting it up as hydriodic acid does in the laboratory, into carbonic acid, ammonia, and glycocine. Is this, then, the explanation of my results?

It seems to me that all the facts I have mentioned above are against any such explanation; for if the minus excretion of urate is due to its destruction, so that it is removed and eliminated in other forms, there ought not to be a rebound, and yet with these drugs the phenomena of the rebound are extremely well marked. So sure as the dose of iodide produces a diminished excretion of urate and a diuresis as its first action, so will this, in turn, be followed by a corresponding plus excretion of urate (see Fig. 1, January 7th and 10th, Fig. 2, October 18th, and Fig. 4, June 24th), with scanty urine, headache, depression, and other signs of its excess in the blood.

But if the iodides do not destroy uric acid, but diminish its excretion by hindering for a time its solubility in the blood, and so prevent its being brought to the kidneys, how do they bring about such a result?

I am afraid that I cannot completely answer this question, but must content myself with pointing out the direction in which a complete explanation should probably be looked for.

As the iodides diminish the excretion of uric acid, we may group them along with all the other drugs that have a similar action, and the chief of those are:

Acids; both vegetable and mineral;

Many salts of the mineral acids;

Acid salts of phosphoric acid;
Salts of nitrous acid;
Sulphur, sulphites, and hypo-sulphites;
Opium, morphine, and cocaine;
Salts or oxides of calcium, lithium, iron, lead, mercury, zinc, copper, silver, manganese, and other metals.

Now everyone of these things, if given by the mouth and absorbed into the blood, diminishes the excretion of uric acid in the urine, and corresponding with such diminished excretion there are relaxed arterioles, lowered arterial tension, and diuresis; and this first action is in all cases also followed after more or less interval by a rebound, with a plus excretion of urate, contracted arterioles, and diminished urine, just as we have seen to occur with the iodides.

Further, it is possible to throw the above-named drugs into groups according to the mode in which they produce their most important effect, the diminished excretion of urate.

In Group I we have the substances which form insoluble compounds with uric acid, such as the metals whose urates are insoluble, as iron, lead, mercury, zinc, copper, silver, manganese, calcium, and others (see 'Uric Acid' pp. 23—40).

Group II.—Those substances which remove or throw out of action one of the solvents of urate naturally present in the blood, as the salts of lithium.

These, as I have shown from the work of others, form an insoluble triple phosphate with the neutral phosphate of soda, which is one of the solvents of uric acid naturally present in the blood; by this means they diminish for a time the solvent power of the blood for uric acid, and as it now holds less in solution, the excretion in the urine is correspondingly diminished ('Trans.,' vol. lxxi, p. 286, and 'Uric Acid,' p. 29).

Group III.—Those which affect acidity, that is, those which raise the acidity of the urine, and probably at the same time diminish the alkalinity of the blood.

We shall see presently that the action of this group is practically identical with that of Group II; but it is nevertheless useful to distinguish them.

This is also the most important group of all, because in physiology the fluctuations in the excretion of urate are practically entirely due to fluctuations in acidity. It includes:

Acids and salts of the mineral acids;
Acid phosphate of sodium;
Salts of nitrous acid;
Sulphur and certain compounds;
Phosphorus and certain compounds;
Opium, morphine, and cocaine.

It appears to me to be extremely probable that the action of the whole of this group depends on the fact that the neutral phosphate of sodium (Na_2HPO_4) is a good solvent of uric acid, and a natural constituent of the blood, while the acid phosphate of sodium is not a solvent (see my paper on "Phosphate of Sodium" in the 'Transactions' of the Society, vol. lxxii, p. 403).

It further appears probable that the neutral phosphate in the blood is present in a condition in which the addition of a quite minute amount of an acid or an acid salt may prevent its acting as a solvent of uric acid at all (see the effects of sulphate of soda in the paper just mentioned).

It appears, then, that any members of Group III may, by diminishing the alkalinity of the blood, interfere with the action of one of its natural solvents of urate, and thus diminish the amount it can hold in solution, and consequently the amount it can bring to the kidneys for excretion; they will thus relax the arterioles throughout the body and lower arterial tension.

With regard to the action of the nitrites, the researches of Professor Leech ('Croonian Lectures,' 1893) seem to show that these substances undoubtedly exercise a direct depressant action on muscular tissue. They also show, however, that these compounds are decomposed in the stomach, giving off nitrous acid, which is absorbed

into the blood and importantly affects its composition and reaction. Thus Professor Leech says ('Lancet,' vol. i, 1893, p. 1502), "When absorbed it [nitrous acid] is no doubt at once converted into an alkaline nitrite, for it can replace carbonic acid in its sodium compounds and convert normal sodium phosphate into acid phosphate, sodium nitrite being at the same time formed."

Now the result of this conversion of normal phosphates into acid phosphates is a rise in the acidity of the urine, which I in common with others have pointed out that the nitrites produce (see 'Uric Acid,' p. 39); and as the normal phosphate of sodium is a good solvent of uric acid, while the acid phosphate is not a solvent at all (see 'Transactions' of the Society, previous reference) the blood becomes, as a result of this action of the nitrites, a bad solvent of uric acid, and ceases to hold it in solution, and as a result of this the arterioles will be relaxed, and arterial tension reduced.

It seems probable, then, that the action of the nitrites on arterial tension is the resultant of two forces: first of all, of the muscular depressant action, weakening the muscle fibre in the heart and the vessel walls generally, an action similar to that of tobacco, lobelia, and other depressor-motors (see 'Brain,' spring and summer number, 1893, p. 245); and second, of the general relaxation of arterioles which all substances produce which clear the blood of uric acid.

The action of most of the other drugs mentioned above will be found to be pretty fully discussed in my book on uric acid and elsewhere, and I need not go into it again here.

Under which of these groups, then, may we place the iodides? Not under Group III, as there is nothing to show that they cause any rise of acidity, and not under Group I, as they are not known to form insoluble compounds with uric acid.

It seems more probable, then, that, like the salts of lithium, they may directly or indirectly interfere with the

solvent powers of the neutral phosphate of sodium, but so far as I am aware there is not at present any direct evidence that this is the case.

Let us now, in conclusion, take a rapid survey of the effects of iodides in health and disease, and see how far their observed action on the excretion of urates will enable us to explain these effects.

In health, iodides produce relaxation of arterioles and diuresis. I have shown in the above figures that the diuresis they produce is contemporaneous with a greatly diminished excretion of urate, and I have further pointed out that some fifteen to twenty or more drugs which have a similar effect on the excretion of urates produce also a similar dilatation of arterioles and a diuresis. Further, I have explained this by suggesting that the effect is in all cases due to the action of urates on the vessels, namely that excess of urate in the circulation contracts the arterioles and capillaries throughout the body, while its more or less complete absence from the blood allows these same vessels to dilate freely, so that the action of any drug is entirely secondary and indirect, and its effects in any given case depend as much on the amount of urate it meets with as on anything else, and if the urate has been previously removed the usual effect fails to appear (see in the case of opium 'Uric Acid,' p. 33).

As regards disease, looking to the fact that iodides clear the blood of urates, and, as a result of this, relax the arterioles and lower tension, it is not at all extraordinary that they should have been found to relieve the uric acid headache (see Dr. Liveing, 'Megrim and Sick-headache,' p. 440), and I have myself found that the best way of treating the headache which iodides produce by way of rebound is to repeat the dose; and the same thing holds for opium, mercury, and other members of the group. Iodides also, in my own case, produce very decided well-being at the time of the diuresis, and in this also they are paralleled by all the other drugs which affect the excretion of urates in the same way. I have also found them very

useful in the treatment of mental depression associated with high tension pulse.

Looking also to the way in which all the members of this group of drugs dilate the arterioles all over the body, it is not to be wondered at that iodides should have been found to promote the healing of skin wounds[1] and the removal of numerous skin eruptions, in all of which they parallel in a remarkable way the action of another drug in the above groups, namely opium.

I may mention also in passing that quite a number of skin diseases are, I believe, due to the evil effects on the nutrition of the skin, of arterioles and capillaries contracted by uric acid; but there is no space to go further into this matter here (see 'Uric Acid,' pp. 121-2 and 251).

In exactly the same way we can understand the iodides being useful in various neuralgias which are so often associated with excess of uric acid in the blood and high tension, and I quite believe in their value in epilepsy, in many besides the syphilitic cases (see Dr. Liveing, prev. ref., p. 443), though, if my experience is of any value, the fits are likely to be worse again when they are left off. The salts of zinc and silver in Group I have been similarly used in epilepsy, and no doubt acted in the same way; and Dr. Liveing remarks with regard to the treatment of headache by valerianate of zinc, that the zinc alone without the valerian has been found to be useful in a variety of troubles of the above nature.[2]

As to asthma, the good effects of iodides are well known, and I believe that this is due to their action on the arterioles. Fig. 4 shows the effects of Hair's cure in myself; no doubt on June 22nd and 23rd there was great relaxation of arterioles, of which the diuresis was but one of many signs (see also my researches in the 'Lancet' of July, 1892, and papers on asthma in 'International Clinics,' 1893).

Again, iodides are recommended by Professor Semmola

[1] 'Lancet,' 1891, vol. ii, p. 831.

[2] Prev. ref., p. 449.

in the treatment of chronic Bright's disease, and have been found to be useful in albuminuria (see Dr. Yeo, 'British Medical Journal,' 1888, vol. i, p. 68); and if albuminuria is due, as I have suggested ('Uric Acid,' pp. 123 and 208), to the arterioles of the skin, liver, and other organs being contracted by urate, there is not much difficulty in explaining their mode of action; and other members of these groups, as acids and opium, have, in my hands, been of use in exactly the same way.

Lastly, of late years we have heard a good deal from Professor G. See and others about the effects of iodides in reducing arterial tension, and the relief from pressure and strain which they may thus afford to a damaged heart; and in the 'Practitioner' for October, 1891, Dr. Lauder Brunton, in an interesting article on "Cardiac Pain and Angina Pectoris," says, after speaking of the use of iodide of potassium in doses of 5 gr. to 30 gr. three times a day, "Whether this acts as a simple eliminant, or whether it tends to increase the blood-supply to the heart by causing absorption of the deposits which block the coronary arteries, or whether it acts in some other way, we cannot at present tell, but about its practical use there can be no doubt whatever."

I think we are now in a position to point out pretty definitely the way that iodides do good in angina, and several other drugs mentioned in my above groups, such as morphine and the nitrites, are also useful in the same conditions, and doubtless act as I have shown in the same way that the iodides do, by clearing the urates out of the blood and allowing the arterioles to relax (see 'Uric Acid,' pp. 32 and 39).

We see, then, that iodides produce a diminished excretion of urates in the urine, and that they do this by directly or indirectly diminishing the power of the blood to hold urates in solution.

Contemporaneously with the diminished excretion of urate so brought about, there is a plus excretion of water

(diuresis) which illustrates the law that the urinary water varies from day to day and from hour to hour inversely as the uric acid excreted along with it; and this again depends on the more important law that arterio-capillary contraction throughout the body, and the arterial tension it produces, vary directly with the amount of urate that is circulating in the blood.

It comes to this, then, that iodides by clearing urates out of the blood allow the arterioles and capillaries all over the body to relax; and this is the secret of all their most important effects in the treatment of disease.

We have seen also that a very large number of drugs mentioned in the above groups have a precisely similar effect on the excretion of urates, and as the result of this an identical effect on the arterioles and capillaries and the excretion of water.

We can now completely understand why iodides, by relaxing the arterioles and lowering blood-pressure, are so useful in reducing the labour of the heart muscle in morbus cordis, Bright's disease, and other conditions associated with high tension; why they relieve headache, epilepsy, and mental depression in cases where the high blood-pressure is interfering with the intra-cranial circulation (see 'Brain,' prev. ref.); why they remove skin diseases and aid the healing of wounds by improving the circulation in the affected parts; and why they diminish or remove albuminuria by freeing the circulation and improving the metabolism in such important organs as the liver, the muscles, and the skin.

On the 27th October, 1891, I read a paper before this Society on "Uric acid as a cause of high arterial tension and its consequences."

In this present paper I have brought forward further facts bearing on the same subject, and have pointed out especially that all these drugs and disease processes which diminish the excretion of urates in the urine do at the same time relax the arterioles, and produce diuresis and other effects of that relaxation and the consequent lower-

ing of arterial tension; that another series of drugs and disease processes, which increase the excretion of urates in the urine, do at the same time, while the increased excretion lasts, contract the arterioles and raise arterial tension, producing a diminished excretion of urinary water and other signs of that contraction of arterioles; and, lastly, there is the fact of the inverse ratio of water and urates in the physiological excretion of urine.

It seems to me that it is impossible to explain these facts except by supposing that urates in the blood stream contract the arterioles and capillaries all over the body, and so produce high arterial tension and its consequences.

I have now shown that the iodides which are known to reduce arterial tension produce at the time of the reduction a diminished excretion of urates in the urine, and that like mercury, opium, and many other drugs a great part of their utility in medicine and surgery is due to their action on the arterioles and capillaries, which again is due to their influence on the solubility and excretion of urates.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 46.)

CASES TO ILLUSTRATE THE RELATIONSHIP

WHICH EXISTS BETWEEN

WRYNECK AND CONGENITAL HÆMATOMA OF THE STERNO-MASTOID MUSCLE.

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It is not my wish, gentlemen, to draw your attention this evening to a consideration of the causes of congenital wryneck. I desire to place on record the cases of hæmatoma of the sterno-mastoid muscle which have come under my notice at the Victoria Hospital in the course of the last five years. Many such cases have already been recorded, so that the mere enumeration of mine would add but little to the general stock of our knowledge. My object in reading this paper before you is to show that a certain relationship exists between wryneck and congenital hæmatoma of the sterno-mastoid.

Congenital hæmatoma of the sterno-mastoid muscle has long been known as an affection of no uncommon

occurrence in children; but nearly all writers upon the subject have occupied themselves with its causation and pathology rather than with the results to which it leads. In all English text-books which I have examined, allusion is made to the fact that injuries received during birth may lead to wryneck; but except in Mr. Owen's book on 'Diseases of Children,' and in a very excellent paper by Mr. Clutton, to which I shall presently refer in greater detail, I have been unable to find any account of cases of hæmatoma of the sterno-mastoid followed out to their termination in wryneck. This omission is no doubt due in part to the difficulty of keeping cases under observation in a large town—partly to the fact that the patient often comes under the care of two different practitioners, first the accoucheur, and then the surgeon to a children's hospital. The main cause, however, is an error which has crept into our ideas of the pathology of the condition; for even so lately as last year Mr. Clutton, in writing about cases of this nature, was obliged to prove that the tumour was not the result of syphilis. If it were more generally recognised that the condition is always due to injury, and to injury alone, the proper pathological term of hæmatoma would be employed, rather than the old and indefinite expressions, "chronic induration" and "tumour" of the sterno-mastoid muscle, which hitherto have been used when alluding to this affection—expressions which have survived in clinical use from the pre-scientific age of morbid anatomy.

In Germany and France, however, where the true cause of hæmatoma has long been known, there has been an equal difficulty in tracing out these cases to their occasional termination in wryneck. Stromeyer, following Dieffenbach, the father of orthopædic surgery, taught that congenital wryneck was almost without exception due to injury done to the sterno-mastoid muscle at birth, and that it was the result of shortening at the place of injury by scar-contraction. This teaching I hope to show this evening is too catholic; but, on the other

hand, it is equally incorrect to assert, as do Petersen and Weiss, that congenital torticollis is never caused by direct injury to this muscle.

Professor Witzel (34),[1] of Bonn, has recently published a paper, "Ueber die Entstehung des sogenannten angeborenen musculären Schiefhalses," in which with true German industry he traces the different views which have been held as to the cause of congenital wryneck from the time of Alexander the Great—who is said to have had a slight torticollis—up to the present time. In this article he discusses the views held by Dieffenbach, Stromeyer, Bohn, Petersen, and others, but he can only quote a few isolated instances in which hæmatoma of the sterno-mastoid resulted in wryneck, because so few cases have been followed to their termination. In Belgium, Tordeus (29), who has written a paper on the subject of hæmatoma of the sterno-mastoid, has not met with better success.

Mr. Bryant (4), in his Lettsomian Lectures in 1863, first drew public attention in England to cases of congenital tumour of the sterno-mastoid muscle, though isolated cases had previously been recorded by Dr. Wilks (31) and Sir James Paget (18).

In 1871, Mr. Thomas Smith (26) read a paper before the Clinical Society of London "On the nature of the so-called Congenital Tumour of the Sterno-mastoid Muscle." He gave an excellent account of the condition, and quoted two cases. The paper is remarkable because he explained correctly the cause and the pathology of the condition, although no post-mortem examination of a similar case was known to him. It is certain that if proper attention had been bestowed on this paper, many children would have escaped a course of grey powder and mercurial inunctions. Mr. Smith makes no allusion to the subsequent occurrence of wryneck.

Mr. Henry Arnott (1), in 1874, records eight cases of this

[1] The reference numbers relate to the table of Bibliography, pages 150 and 151.

affection in a paper entitled "Jottings from the Surgical Out-patient Room." He there states that he has never been able to trace any wryneck in the adult to this condition in infancy, nor has he ever been able to satisfy himself as to the cause of the affection. He does not think it is the result of syphilis or injury, but supposes that the induration is due to some hypertrophic change in the connective tissue occurring during intra-uterine life, which gradually subsides after birth.

Dr. Frederick Taylor (28) had the good fortune to obtain a post-mortem specimen of a sterno-mastoid muscle affected in this manner.

Dr. Spencer (27), however, shows that it was not absolutely the first morbid preparation, as autopsies had been made in similar cases by Heusinger[1] at Würzburg as early as 1826, and by Professor Skrzeczka (25) of Berlin in the year 1869. Dr. Taylor's case is well known. It is published in the 'Transactions of the Pathological Society' for 1875. From his account, and from the drawing of the microscopical appearances which accompanies it, there can be no doubt that in this case the affection was due to a rupture of the muscular fibres. Dr. Taylor does not make any mention of wryneck as a possible consequence of this injury to the muscle, although in his remarks on the case he appears to imply that such a termination is possible.

Dr. Rolleston's courtesy enables me this evening to show you a specimen, which I believe to be almost exactly similar to that obtained by Dr. Taylor. It consists of the greater portion of the left sterno-mastoid muscle of a male child aged one month. The lower tendinous part of the muscle contains a firm and homogeneous white tumour measuring one inch lengthwise and three-eighths of an inch across. From the sections which are placed under the microscope you will see that the tumour consists of well-developed fibrous tissue with a few degenerate muscle fibres entangled in the meshes of the connective tissue.

[1] 'Berichte v. d. königl. Zootomischen Anstalt,' 1826, p. 42.

There is no trace of any blood-clot or blood-pigment. The lymphatic glands round the sterno-mastoid were normal in appearance. The history of the child was unobtainable, but it is interesting to find that the patient died from the effects of a hæmorrhage into his right lateral ventricle. The tumour is therefore possibly the result of a hæmorrhage into the muscle during intra-uterine life, for neither in this case nor in that presented by Dr. Taylor is it likely that such complete disappearance of the blood-clot would have taken place in a month.

Mr. R. W. Parker (19) with his usual care and accuracy, has published an excellent series of cases of hæmatoma of the sterno-mastoid muscle which have come under his notice. He says in this paper that "It would be difficult to disprove Mr. Smith's theory as to the cause of the affection," though he does not wholly give in his adherence to it. In Mr. Parker's cases the swelling was confined to the lower and tendinous part of the muscle. This is not, however, by any means an invariable case, for out of a total of 106 cases which I have gathered together from different sources, the swelling was situated in the upper and middle parts of the muscle in twenty-two, in the lower part in thirteen, in one the whole muscle was affected, whilst in the remaining cases the exact position is not noted. In connection with statistics it may be as well to add here that in these 106 cases recorded by English, American, German, French, and Belgian surgeons the right sterno-mastoid muscle was the seat of injury 47 times, the left 36 times, and in five it was bilateral.

Mr. Clutton (6) gives the best clinical account of these cases in the 'St. Thomas's Hospital Reports' for 1888, and I have to offer him my very best thanks for allowing me to use some of them this evening. He gives notes of eighteen cases which have come under his personal observation, and after a very careful examination of the data thus obtained he finds himself in a position to agree with Mr. Smith that violence is the main cause of "tumour

of the sterno-mastoid." This paper is also of especial interest, because Mr. Clutton incidentally draws attention to the fact which I wish to demonstrate this evening, that in some cases—four out of the eighteen enumerated in his series—a tendency to wryneck ensued upon congenital hæmatoma of the sterno-mastoid muscle.

Mr. Golding-Bird (9) in an able paper on congenital wryneck, makes a very necessary distinction between the simpler form of wryneck, which we are now considering, due in some cases to hæmatoma of the sterno-mastoid, and the much more severe form accompanied by facial hemiatrophy, which he thinks may sometimes be produced by an acute local encephalitis.

Finally, Dr. H. R. Spencer (27) has recently completed an interesting investigation into the pathology of "Hæmatoma of the Sterno-mastoid Muscle of New-born Children." In this paper details of fifteen cases are given, in nine of which microscopic sections were prepared. These sections Dr. Spencer has been so kind as to bring here to-night. They prove beyond the possibility of doubt that the cause of the condition is injury resulting in a rupture of the fibres of the sterno-mastoid muscle with effusion of blood into the connective tissue. Dr. Spencer also draws attention to the fact that such injuries may very probably terminate in wryneck, although he is unable to bring forward any cases which have come under his own observation.

I have seen five cases of hæmatoma of the sterno-mastoid muscle during the last five years. Mr. Edgar Willett has kindly allowed me to examine three of his cases, and Mr. Clutton, with equal courtesy, has given me leave to use the notes of some of his unpublished cases.

In none of these cases, so far as I could observe, was there any obvious asymmetry of the face. This point is of great interest, for it appears possible that the presence or absence of facial asymmetry may perhaps serve to distinguish between these comparatively slight cases of wryneck due to injury and the more severe forms which,

as Mr. Golding-Bird has pointed out, may be caused by much more obscure lesions than partial rupture of a superficial muscle. The notes of these cases are briefly as follows :

Case 1.—Ashley T—, a boy æt. 6 months. Hæmatoma of the right sterno-mastoid muscle. Forceps had been applied at birth with sufficient force to indent the right temple. A slight wryneck ensued, which in the course of eighteen months yielded to simple treatment by shampooing. When the boy was last seen he had an alternating squint. There was no trace of the original hæmatoma, but the dent on his right temple was still perceptible. He was an only child.

Case 2.—Frederick W—, æt. $3\frac{1}{4}$ years ; second child. There was an induration of the right sterno-mastoid muscle at the point where it is crossed by the external jugular vein. Breech presentation. The wryneck was sufficiently well marked to render division of both heads of the sterno-mastoid necessary. Five years later the head was evenly balanced, but the induration was still perceptible.

Case 3.—Edith J. B—, æt. $5\frac{3}{4}$ years. Breech presentation. Three weeks after birth a hæmatoma of the right sterno-mastoid muscle was noticed. The child has had a wryneck for the last year. Both heads of the sterno-mastoid were divided. It was noticed at the time of the operation that there was a hard thickening which extended through the muscle about the middle of its long diameter.

Case 4.—Baby B—, a girl æt. 1 month. Hæmatoma of both sterno-mastoids immediately above their sternal heads. First child. Mother aged 33. Very hard labour ; head presentation ; forceps were used. Both swellings eventually disappeared, and the mother says that she does not remember to have observed any ten-

dency to wryneck. The child died of pneumonia at the age of two years and ten months.

CASE 5.—Thomas C—, æt. 6 weeks; ninth child. Head presentation. Easy labour attended by a midwife. Well-marked hæmatoma of the left sterno-mastoid muscle about its middle. Five months later death occurred from tubercular meningitis. There was no tendency to wryneck.

CASE 6.—John H—, æt. 5 months; first child. Hæmatoma of the right sterno-mastoid. Forceps delivery. Death from tabes mesenterica at thirteen months. No history of wryneck could be obtained.

CASE 7.—Frank McG—, æt. 5 months. Hæmatoma of the right sterno-mastoid, first noticed at five weeks. The hæmatoma is in the substance of the muscle just above its sternal head; the tendon can be felt below the tumour and appears to be normal. First child, but the mother has had one miscarriage. Head presentation; forceps were used. There is a slight inclination of the head towards the affected side, but at present it is too slight to call a wryneck. The case is under the care of Mr. Edgar Willett.

CASE 8.—Alice L—, æt. 13 years; has had a wryneck almost since birth. A tumour in the left sterno-mastoid "like a bone" was first noticed when she was a few weeks old. The head was then straight. The deformity came on subsequently, and was eventually so severe as to render tenotomy necessary. There was distinct asymmetry of the face.

CASE 9.—Frederick P—, æt. $3\frac{1}{4}$ years; the ninth child. A lump was observed on the right side of his neck shortly after his birth. He was brought to the hospital to be cured of a wryneck; on examination it was found that

the right sterno-mastoid was rigid and thickened along its whole length. Both heads of the muscle were divided.

CASE 10.—Annie W—, æt. 1 month; first child; mother aged 51; married fifteen years; no miscarriages. Breech presentation; very hard labour; no instruments used. Hæmatoma of the right sterno-mastoid in its lower third. The child keeps its head persistently to the right side.

CASE 11.—Edith H—, æt. 4 months. Sixth child. Transverse presentation. Version. Hæmatoma of the left sterno-mastoid in its lower third. The swelling was first noticed fourteen days after birth. It was getting smaller when the child was last seen, and there was no suspicion of a wryneck.

The following cases have already been published by Mr. Clutton in the eighteenth volume of the 'St. Thomas's Hospital Reports' in the paper to which reference has already been made.

CASE 12.—Harriet C—, æt. 6 weeks. Tumour of the right sterno-mastoid. No evidence of syphilis. Foot presentation. Twelve hours in labour. Great traction was made upon the presenting feet. In four months it is noted that the tumour had "quite gone."

This patient came again as an out-patient at seventeen months, and again at two years of age, with well-marked wryneck from contraction of the right sterno-mastoid. No contraction of the trapezius. Mr. Clutton was about to treat this case by tenotomy when it disappeared from his clinic.

CASE 13.—Charles C—, æt. 2 weeks. Tumour of the left sterno-mastoid at the junction of the upper with the middle third. It was a large tumour, but apparently confined within the sheath of the muscle. No evidence of syphilis. Mother aged twenty-three. First confine-

ment. In labour two or three days. Forceps used. Two months after this baby was first seen, the tumour had almost entirely disappeared, but there was well-marked contraction on that side producing wryneck.

Case 14.—Walter B—, æt. one month. Tumour of left sterno-mastoid at the junction of the middle with the upper third. Long fusiform swelling with soft centre; left muscle shorter than the right. Right clavicle deformed from recent fracture; first noticed two days after birth. No evidence of syphilis. Mother aged thirty. Has had four children. Was in labour on this occasion two or three days. Head presentation. "They all had a turn at the child's head." "Dragged away at last," but no instruments were used. In two months the tumour had almost disappeared, but there was a suspicion of commencing wryneck. Mr. Clutton did not see the child after this note.

Case 15.—Alfred P. J—, æt. five weeks. Tumour of right sterno-mastoid, upper half. In part fluctuating; accompanied by contraction so as to hold the head down on that side. First noticed fourteen days after birth. No evidence of syphilis. Mother aged twenty-two. First confinement. Short labour. Head presentation. Instruments used by doctor.

I find on putting my results into a tabular form that there are 106 cases of hæmatoma of the sterno-mastoid muscle easily accessible in the English and foreign periodicals. In 20 of these cases death occurred too soon for wryneck to have become apparent; in 47 cases wryneck was not looked for; in 25 cases it occurred, but in 4 it was so slight and transient as to be unimportant; in 14 cases wryneck was looked for but was not found. Well-marked wryneck therefore occurred in 21 cases at least out of the total of 106 cases of congenital hæmatoma which are recorded. In Germany Dr. Bohn

(2), of Königsberg, and in France Dr. Rédard (23) have endeavoured to ascertain whether hæmatoma of the sterno-mastoid leads to torticollis. Dr. Bohn thinks that such a termination is possible, but Dr. Rédard believes that it is most unusual. In England, however, the observations of Mr. Clutton, of Mr. Edmund Owen, and of myself are conclusive. We have actually traced out cases of hæmatoma of the sterno-mastoid muscle from early infancy to the time when they required tenotomy of the muscle for the relief of wryneck. Mr. Owen's case was an isolated one, so it need not be further considered. Taking Mr. Clutton's, Mr. Willett's, and my own together the total number is thirty; of these eleven eventually had wryneck, but in two of the eleven cases the torticollis was so slight that it was hardly worth while taking it into account, whilst in four cases it was necessary to divide the tendon.

"In the present day," Cardinal Newman wrote, in the first volume of his essays Critical and Historical, "mistiness is reputed the mother of wisdom." I do not wish to be esteemed wise, and I will therefore avoid being misty by putting my conclusions as concisely and plainly as possible.

It appears that—

1. Hæmatoma of the sterno-mastoid muscle may be due to intra-uterine injuries, which are rare, or to injuries at the time of birth, which are common.

2. Congenital hæmatoma of the sterno-mastoid muscle occasionally predisposes to wryneck in children who are otherwise healthy.

3. Wryneck due to hæmatoma of the sterno-mastoid varies in degree from the slightest and most transient form to one of such severity as to require tenotomy of the muscle for its cure. The wryneck thus produced is not —so far as I have seen—accompanied by asymmetry of the face.

4. Every child who has the misfortune to be injured in this manner must be carefully watched for a much longer

period of time than is usually considered necessary to obviate any tendency to the formation of a wryneck.

5. Surgeons should give more prolonged attention to children affected with hæmatoma of the sterno-mastoid muscle to enable them to determine still more accurately what are the ultimate and usual effects of the injury.

I believe that these conclusions are trustworthy, for care has been taken not to base the premises on any selection of cases. The subject is an interesting one, although it is only of minor importance in surgery, and I trust that you will not feel your time has been ill-spent in listening to this account of it.

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(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 52.)

ON THE

ABSENCE OF SUGAR FROM NORMAL HUMAN URINE.

BY

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THE hypothesis that normal human urine contains sugar has on the whole been favoured more by chemists than by physiologists. The reason of this is that the chemical methods employed for detecting sugar depend for the most part upon the reducing action of that substance; and, until a comparatively recent date, no test for sugar was in common use which did not give more or less reaction with normal urine. Therefore, in the absence of a sufficient knowledge of other reducing agents in urine which react in the same way as sugar with the tests employed, it was concluded that sugar was invariably present in that secretion in small quantities.

I have convinced myself that sugar is absent from human urine by two methods:

1st. By studying the amount of reduction which normal urine produces; by isolating the ingredients of normal urine which effect reduction in the same way as sugar, estimating their amount and their reduction equivalents; and—

2nd. By applying to human urine a most sensitive test for sugar, with *which that secretion gives absolutely no reaction.*

I will first give a brief sketch of the methods I have employed.

1st. Methods for isolating the reducing agents of normal urine, which resemble sugar in their action upon cupric oxide in boiling alkaline solutions.

Having observed the invariable presence of reducing agents in normal human urine, every specimen of which is found to exert more or less reduction of copper oxide in boiling alkaline solutions, I endeavoured to ascertain the nature of the substance or substances to which this reaction is due by the method of *precipitation.* The details of this investigation are published in the 'Proc. Roy. Soc.,' vol. xliii, pp. 493—534. Briefly the results obtained are as follows.

Precipitation with *mercuric chloride* removes from normal urine all substances which are capable of effecting reduction of copper oxide in the same way as glucose, but any glucose which may be present in diabetic urines is entirely unaffected by this treatment.[1] The proof that the reducing agents are removed by this method is that if the excess of mercury salt be subsequently precipitated by ammonia, the filtrate is destitute of reducing action. There are two substances in normal urine which resemble sugar in their reducing action upon cupric oxide: one a highly nitrogenous acid—*uric acid;* the other a highly nitrogenous base—*kreatinin.*

[1] After adding a knowu quantity of glucose to a normal urine, then precipitating completely with mercuric chloride, I have found the glucose unaltered in the filtrate.

When mercuric chloride is added to urine the uric acid is precipitated immediately as mercury salt, and may be separated by immediate filtration; the kreatinin, however, is deposited very gradually from the filtrate in the form of a spherical mercury salt, forty-eight hours being required for complete precipitation.

About one-fourth of the reduction effected by normal urine is due to the uric acid which it contains, three-fourths to the kreatinin.

By weighing the mercury salt of kreatinin passed in twenty-four hours, analysing it, and finding that it contains about 20 per cent. of kreatinin, I have computed the daily output of this base by a healthy man at from 1·8 to 2·1 grammes in twenty-four hours.

Further, I have found that 12 grammes of this urinary kreatinin (a beautifully crystallised specimen of which I have brought for your inspection) are equivalent in reducing power to 10 grammes of glucose.

If we take 52·8 fluid ounces as the volume of urine passed in twenty-four hours, it follows that the reduction of copper oxide effected by the kreatinin of the urine is equivalent to that which would be produced by from 0·43 to 0·51 grain of glucose per 1 fluid ounce.

But the total reduction effected by normal human urine has been estimated as equivalent to 0·6 to 0·8 grain of glucose per fluid ounce, and one-fourth of this is due to uric acid; so that the entire reduction effected by the normal secretion is accounted for by these two substances.

My observation that one-fourth of the reduction effected by normal urine is due to uric acid confirms the work of Dr. F. W. Pavy, F.R.S. (vide 'Med.-Chir. Transactions,' vol. lxiii, p. 222). He found that precipitation of normal urine with lead acetate removed one-fourth of the reducing agents. I observed that the reduction of copper oxide by a normal urine always exceeds that effected by the same specimen upon picric acid (as applied by my father, Sir George Johnson), the ratio being as

4 : 3 ; and this discrepancy was traced to the fact that picric acid is not reduced by uric acid in boiling alkaline solutions, but it is reduced by the kreatinin.

As regards the reducing action of the urinary kreatinin, it was suggested by Dr. Oliver, in his 'Bedside Urine Testing,' that this base was responsible for much of the reduction effected by normal human urine upon cupric oxide.

Kühne also draws attention to the reduction of cupric oxide by kreatinin in boiling alkaline solutions in his 'Lehrbuch der physiologischen Chemie,' p. 505.

But the *amount* of reduction attributed to kreatinin by German workers is small ; thus E. Salkowski, in 'Centralblatt für die medicinischen Wissenschaften,' March, 1886, estimates the reduction due to uric acid and kreatinin taken together as varying from one-fifth to one-sixth of the total cupric oxide reduction effected by normal urine.

It remains, therefore, for me to explain the much greater reduction which I have observed in the kreatinin isolated by myself.

In its chemical relations kreatinin is closely allied to the vegetable alkaloids. The substance is, in fact, an animal alkaloid.

It is well known that substances of complex constitution are peculiarly liable to undergo isomeric changes under the influence of chemical agents, and especially when aided by heat; so that after a lengthy and prolonged method of extraction has been resorted to, it does not by any means follow that the substance ultimately isolated is identical with that originally present.

I attribute the differences (which are many) between the kreatinin isolated by me from human urine, and the kreatinin prepared and isolated by other observers, to the fact that all through the series of processes which I have made use of, not only are violent chemical agents avoided as far as possible, but especially no heat is applied from first to last, so that I look upon my kreatinin as the actual base present in the urine—unchanged during extraction.

I think that this point will be made clear by a glance at the subjoined table from the 'Proc. Roy. Soc.,' vol. 1, p. 291.

Methods for Isolation of Kreatinin from Urine.

I. Heintz and Pettenkofer.	II. Liebig.	III. Maly.	IV. The Author.
Fresh urine neutralised with sodium carbonate, evaporated to a syrup. Syrup exhausted with alcohol, and alcoholic zinc chloride added.	Fresh urine neutralised with milk of lime. Calcium chloride added to complete precipitation. Filtrate evaporated till the salts crystallise out. Zinc chloride added to the liquor. Kreatinin zinc chloride dissolved in boiling water and treated with lead hydrate at the boiling temperature. Filtrate evaporated.	Urine evaporated to $\frac{1}{3}$ of its original bulk. Lead acetate added. Filtrate freed from lead by H_2S. Filtrate neutralised by sodic carbonate and precipitated by mercuric chloride. Precipitate suspended in water, and decomposed by H_2S. Filtrate evaporated. Residue recrystallised from alcohol.	Fresh urine + $\frac{1}{10}$ of its volume of cold saturated solution of sodic acetate, + $\frac{1}{3}$ of its volume of cold saturated solution of mercuric chloride. Filter immediately. Collect the precipitate which forms in the filtrate in 48 hours. Decompose the Hg salt by H_2S under water. Treat filtrate with $Pb(HO)_2$ at ordinary temperature. Evaporate filtrate *in vacuo* over H_2SO_4.
	Product, kreatinin mixed with kreatine.	Product, kreatinin hydrochloride.	Product, efflorescent urinary kreatinin.

Differences between the kreatinin isolated by Author and the kreatinin described by Liebig:

	Kreatinin of urine.	Kreatinin (Liebig).
Solubility in water .	1 in 10·78 at 17° C.	... 1 in 11·5 at 16° C.
„ in alcohol	1 in 362 at 17° C.	... 1 in 102 at 16° C.
Platinum salt .	$2(C_4H_7N_3O.HCl).PtCl_4 2H_2O$	... $2(C_4H_7N_3O.HCl).PtCl_4$.

The difference in reducing action is not more remarkable than some of the differences recorded in the above table.

So far I consider that the absence of sugar from normal human urine is proved, inasmuch as the entire reduction which the secretion can effect has been accounted for by two definite substances which have been

isolated from it, their amount therein ascertained, and their reducing power determined—viz. uric acid and kreatinin.

But an additional proof is afforded by the application of an extremely sensitive test for sugar, which gives no reaction whatever with the normal secretion of the human kidney; and this brings me to my second point.

2nd. The phenylhydrazine test for sugar.

C. Schwartz ('Pharm. Zeit.,' xxxiii, 465) describes the following test, which, though extremely sensitive in reaction with sugar, gives absolutely negative results with normal urine.

The urine is first precipitated completely with lead acetate and filtered, the filtrate is mixed with a solution of phenylhydrazine and excess of potassium hydroxide, and boiled for a minute. If sugar is present a yellow colour appears, followed by an orange precipitate on adding excess of acetic acid. The formation of this precipitate is more significant than the development of colour.[1]

When this test is applied to normal urine no indication of sugar is obtained, and even the preliminary precipitation by lead acetate is unnecessary. For clinical purposes, however, it is better to precipitate first with lead acetate, since a urine loaded with urates may give a precipitate with acetic acid, which might be mistaken for the sugar precipitate. This is, I take it, the only reason for precipitating with lead acetate before applying the test, for uric acid gives no reaction with the phenylhydrazine.

It will be seen that we have now three tests for sugar in urine, which react as follows:

(1) The cupric oxide test reacts with—

(*a*) Uric acid.

(*b*) Kreatinin.

(*c*) Glucose.

[1] The reason of this is that boiling with potassium hydroxide slightly deepens the colour of normal urine.

(2) The picric acid test with—
  (*a*) Kreatinin.
  (*b*) Glucose.
(3) The phenylhydrazine test responds with—
  Glucose only;

and the last gives absolutely no indication with normal urine, thus proving the absence of sugar from that secretion.

To ascertain whether anything present in normal urine interferes with the delicacy of the phenylhydrazine test, I have added known quantities of glucose to the secretion, and found that I could easily detect $\frac{1}{20}$ of a grain of glucose in one fluid ounce of urine.

In using the phenylhydrazine test for sugar, the crystalline phenylhydrazine hydrochloride should be employed in the proportion of four times the weight of the sugar present. In practice, as much as will lie on the end of a penknife is sufficient for one fluid drachm of urine. If sugar is present, even in so small a proportion as $\frac{1}{20}$ of a grain to the fluid ounce, the precipitate after acetic acid appears *within ten minutes.*

Finally I may refer to the clinical importance of the above facts. It is common, especially in the summer months, to meet with specimens of urine of high specific gravity (1030 and upwards), which give decided reduction of copper oxide, perhaps equivalent to from 1 to 1·5 grains of glucose per fluid ounce. Yet such urines are usually quite free from sugar. They are instances of so-called azoturia; mixed with nitric acid and stirred, they generally deposit abundant crystals of urea nitrate; and the other nitrogenous excreta, uric acid and kreatinin, being proportionately abundant, account for the increased reduction of cupric oxide. In such cases the phenylhydrazine test gives no indication of sugar, and at once proves the absence of that substance.

Appendix.

After an examination of the reaction of carbo-hydrates with benzoyl chloride, recommended to my notice by Dr. W. Hunter during the discussion on my paper, I see no reason to modify my statements as to the absence of glucose from normal human urine.

It is true that benzoyl chloride produces a precipitate when shaken with normal urine in presence of caustic soda, but as the test gives apparently identical results with glucose, starch, and many other non-saccharine carbo-hydrates, and as the phenylhydrazine test for sugar is so extremely sensitive, I consider that there is no reason to believe that normal human urine contains sugar.

Indeed, as a result of careful experiments conducted since my paper was read, I find that the phenylhydrazine test would detect sugar in the urine even if the quantity present did not exceed $\frac{1}{100}$ of a grain per fluid ounce.

Of course, for ordinary clinical testing, such delicacy of observation is unnecessary, the copper and picric acid tests being sufficiently sensitive for all cases.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 59.)

TWO CASES

OF

KNEE TROUBLE WITH DIFFICULTY IN LOCOMOTION,

DEPENDING UPON AN ELONGATION OF THE LIGAMENTUM PATELLÆ,

TREATED BY TRANSPLANTATION OF THE TUBERCLE OF THE TIBIA.

WITH REMARKS.

BY

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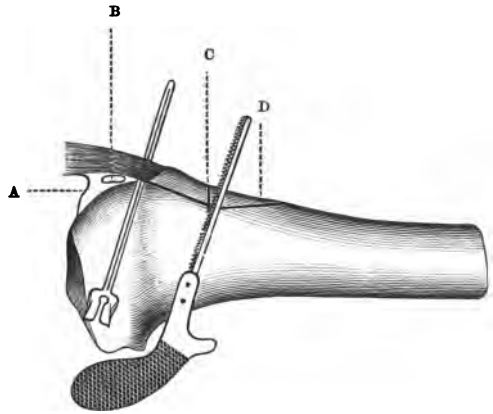
Received December 12th, 1892—Read February 14th, 1893.

Case 1.—C. W—, a housemaid, æt. 21, was sent to the Orthopædic Department of St. Bartholomew's Hospital by Dr. Collins, of Sawbridgeworth, for trouble in the knee-joints and difficulty in locomotion. Whilst walking, some six years ago, her left knee, as she expressed it, suddenly gave way, causing her great pain, and subsequently swelling up. Three months later her right knee was affected without apparent cause in the same manner. The swelling of the joint soon subsided, but the knees remained weak and always painful on walking, especially

on uneven ground. Her patellæ suddenly and without warning would slip round on to the outer or inner condyle, locking the joint, and giving her great pain. At times she would actually fall, dropping whatever she might be carrying. She was thus quite incapacitated for her duties as a housemaid. The knees at first sight appeared normal, but on examination the ligamentum patellæ was found so elongated that the patella could be pushed much higher than natural on the anterior surface of the femur, and could be completely dislocated either inwards or outwards on to the corresponding condyle. When the knee was flexed the patella could be made to ride on the lower end of the femur, so that its anterior surface looked upwards instead of forwards. In other respects the knee-joints appeared normal. The patient had worn an elastic knee-cap without any benefit. As the disability in the knees appeared to depend upon this elongated condition of the patellar ligament, it was decided to shorten it by transplanting the tubercle of the tibia. On June 16th, the left leg having been previously prepared in the usual way for an aseptic operation, a longitudinal incision, four inches in length, was made over the ligamentum patellæ and tubercle of the tibia. The ligament at its insertion into the tubercle having been freed, a director was passed beneath it and left *in situ*. Two lateral incisions having been carried downwards through the periosteum on either side of the tubercle, a transverse incision with the saw was made immediately below the tubercle for about half an inch into the bone, and one inch below this a second incision was carried upwards and backwards obliquely through the bone into the bottom of the transverse incision as shown in the diagram. A triangular piece of bone was thus removed from the tibia below the tubercle. This enabled a keyhole saw to be applied below the tubercle and to be worked upwards through the bone towards the director beneath the ligamentum patellæ.

The tubercle with the ligamentum patellæ was thus

detached, leaving the periosteum on its anterior surface undisturbed. A bed was next made for the tubercle on the anterior surface of the tibia, one inch lower than its normal situation, by chiselling out and thus levelling sufficient of the bone from the already obliquely sawn surface.



The lines of incision for transplanting the tubercle of the tibia in the operation of shortening the patella ligament. A. Synovial membrane of knee-joint. B. Bursa under patella ligament. C. Transverse vertical incision. D. Oblique incision.

The tubercle was now easily drawn down into its new situation, and fixed by an ivory peg. To prevent any slipping upwards a ridge was left by slightly deepening the sawn surface of the tibia. The patella could now no longer be dislocated on to the outer and inner surface of the respective condyles, nor be pushed up as before on to the anterior surface of the femur. The skin-wound was united accurately without drain, and the knee well covered with sal-alembroth gauze, firmly bandaged, and immobilised in the extended position by a plaster-of-Paris bandage reaching from the ankle to above the middle of the thigh.

The patient had no bad symptom; the plaster case was not removed nor the wound dressed till July 19th, when the wound was found soundly healed. A leather splint

was moulded firmly to the limb, and the patient left the hospital for her home on July 18th. When last seen in October, 1892, she had free movement of the joint, and the patella could no longer be dislocated laterally or pushed upwards. She had no pain or inconvenience of any kind. She is to come into the hospital to have the other leg operated on when sufficient time has elapsed to show that the benefit she has received is permanent.

CASE 2.—M. G—, æt. 17, a nursemaid, was admitted to Sitwell Ward, St. Bartholomew's Hospital, on October 14th, 1892, from the Orthopædic Department. She first came under notice in May, 1892, but was kept waiting before any operation was advised till it was seen how the operation in Case 1 succeeded. The history was similar to that of the first patient. Six years ago she had a fall through stepping on a stone when running down a hill, her right patella appearing to slip over to one side. She subsequently had a series of falls, and noticed that her knee-cap appeared very loose. About three years ago she found that her left knee was in a similar condition. She complained of pain and disability, and of sudden falls without warning if she attempted to walk without a bandage on the knees; but she said she could walk with a bandage three or four miles on level ground, as a rule without trouble. On examination there was found a general laxity of tissues around both knee-joints. The patellar ligament was lengthened, allowing very considerable displacement of the patella to either side, and in the left knee the bone could be pushed so far inwards and outwards as to uncover almost completely the condyles. The patellæ were somewhat smaller than normal. On October 20th the tubercle was transplanted on the left side, three quarters of an inch lower down the tibia, in a manner similar to that described in the first case, the tubercle with the patellar ligament being drawn down till the patella could no longer be laterally displaced. It was then fixed as in the first operation with an ivory peg.

On November 12th the plaster-of-Paris case and dressings were removed for the first time after the operation, and the wound was found to have healed by the first intention. On November 20th the patient left the hospital, wearing for precaution's sake a poroplastic splint. The patella could no longer be displaced, and the knee was to all appearance in a normal condition. She too, like the first patient, is to come back to the hospital later to have the ligament of the opposite knee shortened.

Remarks.—I have ventured to bring these cases before the notice of the Fellows of the Royal Medical and Chirurgical Society; in the first place because, as far as I know, this condition of the knee-joint depending upon an elongation of the ligamentum patellæ has not come prominently under the notice of surgeons; and secondly, because I believe that the operation for shortening the ligament by transplanting the tubercle of the tibia has not previously been described. I find no account of the affection in the text-books on surgery, nor in the special works on joint disease that I have been able to consult. I am under the impression, however, that the condition is not so rare as I at first imagined it to be. Since the first case came under my notice last May I have met with two other similar cases—the one here described as Case 2, and a third a few weeks ago, in which, however, the elongation of the ligament was not so great, and the knee trouble less marked. I find, moreover, that Dr. Shaffer, of New York, in the fourth volume of the 'Transactions of the American Orthopædic Association,' has described seven cases of elongation of the ligamentum patellæ in which the patient had similar symptoms to those mentioned in my cases, and he says that he has met with other cases in which there was knee trouble due to a lengthened anterior patellar ligament. A condensed description of Dr. Shaffer's published cases is appended to this paper. No operative treatment was undertaken by him in any of them. He proposed in one

case the shortening of the ligament, but the patient would not consent. As in my own cases, except in one in which the elongation followed the breaking down of adhesions for stiff knee, no cause for the elongation of the ligament could be discovered. In three there was slight knock-knee; in one marked knock-knee. In none of mine was this condition of the knee present. In one of Dr. Shaffer's cases, and in the right knee of one of my own, the patient attributed the trouble to a fall. I think, however, that the fall was rather the result of the elongation of the ligament allowing the patella to be dislocated, than that the stretching of the ligament was due to the dislocation, since this appears to have been evidently the case as regards the left knee, and in my first case the patient seemed clear that her first fall was the result of the laxity of the ligament, the knee giving way, as she expressed it, before she fell.

In three of Dr. Shaffer's cases a peculiar outgrowth of bone was felt at the intercondyloid notch in the position in which the patella would normally rest. There was no sign of such an outgrowth in any of my cases. The length of the elongated ligament when measured from the top of the tubercle to the lower border of the patella, with the knee bent at an angle of 90°, varied from 2¾ to 3½ inches. The normal ligament when measured in a similar way was found by Dr. Shaffer to vary in the adult from a little over 1 inch to 1½ inches, and in a few tall persons to 2 inches. These measurements agree with my own tables in a similar state of the joint. From the consideration of my own and Dr. Shaffer's cases I think we may generalise as follows:

1. That no efficient cause for the condition is known.

2. That generally one knee first, and the other subsequently, becomes affected.

3. That the affection is more common in women than in men.

4. That it is attended with disability and weakness in

the joint and sometimes pain, especially on walking on rough and uneven ground; difficulty in going up and down stairs; sudden, partial, or complete dislocation of the patella without warning and without apparent reason, generally causing the patient to fall; and recurrent slight attacks of synovitis of the knee.

5. That the patella during extension of the leg can be dislocated on to the outer and inner surface of either condyle respectively, and, with the leg bent at an angle of 90° in the sitting posture, can be pushed up on to the condyles of the femur, so that its anterior surface looks almost directly upwards instead of forwards and slightly upwards.

6. That instrumental treatment is of practically little service, though in some cases it appears to have given relief.

7. That shortening of the patellar ligament satisfactorily deals with the affection.

I was led to transplant the tubercle of the tibia rather than to excise a portion of the ligament and suture the divided ends: in the first place because by doing so there was practically little danger of opening the knee-joint; in the second place, it avoided any risk of the ligament after division failing to unite; and in the third place, because with the tubercle detached the ligament could be drawn down till on manipulation the patella could no longer be dislocated over the condyles, and thus the amount of shortening necessary could be accurately gauged. The ridge cut in the bone immediately above the upper border of the tubercle effectually prevented any subsequent slipping upwards of the tubercle, and consequently of the ligament. The technique of the little operation is described in detail in the notes of the case. I may say in passing that transplantation of portions of bone with ligaments or tendons attached is applicable to several other deformities: for example, I have recently transplanted the tuberosity of the os calcis with the tendo Achillis for overcoming the elongation of the calf

muscles in paralytic talipes calcaneus; and Poncet[1] has also transplanted the tubercle of the os calcis for the purpose of approximating the ends of a ruptured tendo Achillis, and the tubercle of the tibia for bringing together the fragments of an old fracture of the patella. Other examples of cases in which transplantation of like portions of bone might with advantage be undertaken will no doubt present themselves to the Fellows of the Society.

ADDENDA.

Cases published in the Fourth Volume of the 'Transactions of the American Orthopædic Association' by Dr. Shaffer, of New York.

CASE 1.—A man, æt. 25, complained of difficulty of locomotion, and especially of great difficulty in going up and down stairs. He was at first believed to be suffering from some form of paralysis. He had to walk very carefully, or he would upon the slightest lateral jar dislocate one of his knee-caps laterally and fall suddenly to the ground. There was slight knock-knee on either side, and there was present at the intercondyloid notch a very perceptible tumour—an apparent exostosis, which occupied the position in which the patella would normally rest. The patellar ligament measured 3½ inches. With the knees flexed the patellæ were drawn above the condyles of the femur. The patient knew of no cause for his trouble. Several attempts, but without avail, were made to relieve the condition by an apparatus. He finally obtained some comfort by the use of an elastic knee-cap with a lateral rubber pad.

CASE 2.—A lad, æt. 14 years, who had had hip disease and had of late grown rapidly, showed signs of knock-knee. His patellar ligaments were found to measure

[1] 'Revue d'Orthopédie,' July, 1891.

3½ inches on the left side and 3 inches on the right. The upward displacement of the patella was marked during flexion of the knee. There was a distinct tumour on the intercondyloid notch on either side, more marked on the left, and the patient moved up and down stairs with an awkward gait, and fell at times without apparent cause. On one occasion he fell and dislocated his right patella, on another occasion his left. The left patella was not reduced by the surgeon who saw him, and on his return to New York all attempts to reduce it failed. By the use of a leg brace giving lateral support he could walk well, although the left patella remained unreduced.

CASE 3.—A lady, æt. 35, was unable to walk except with great difficulty, and was always in danger of falling. The least irregularity of the pavement would cause a fall. She had never dislocated her patella. There was slight knock-knee. She had great difficulty in going up and down stairs. She was treated by light lateral leg braces which prevented lateral movements of the knee and rotation of the tibia during flexion. From this apparatus she received great relief.

CASE 4.—A girl, æt. 13, two years ago fell on a piece of orange peel and dislocated her left patella. She subsequently dislocated her right. She now falls frequently and easily. After each fall the knee swelled and there was great pain, which gradually disappeared. A knee-cap did no good. The patellar ligaments measured 2¾ and 2⅝ inches respectively. The patellæ were freely movable, and in full extension with the knees at rest lay above the condyles. There was no knock-knee. There was an evident exostosis at the sulcus between the condyles of the femur. She was fitted with leg braces limiting the rotation at the knee. She obtained some relief, but was lost sight of.

CASE 5.—Miss X—, age not stated, had had both knees

for a long time immobilized in plaster-of-Paris dressing, with consequent fibrous ankylosis. The ligament was stretched by the force required to break up the sub-patellar adhesions. The right ligament measured 2¾, the left 2½ inches. "An express disability exists in this case, occasioned partly by the reduced quadriceps power in knee-use, and partly by a disagreeable 'click' which occurs when the patella, especially on the right side, slides into its supra-condyloid position."

CASE 6.—A young man found difficulty in playing lawn tennis; his knees, as he expressed it, "gave out very quickly." He had pain after use of the knee, and heat and swelling were sometimes present. He wore knee-caps without relief. He declined to submit to an operation.

CASE 7.—A lady found difficulty in going up and down stairs. The elongation of the patellar ligaments was very marked. Nothing was done, as the patient rejected the remedies proposed.

Table of Cases of Knee Trouble from Elongation of Patellar Ligament.

Case.	Surgeon.	Sex.	Age.	Cause.	Ligament measured.	Signs and symptoms.	Knock-knee.	Exostosis between condyles.	Treatment.	Result.
1	Shaffer	M.	25	None known	3¼ inches	Difficulty in going up and down stairs. Sudden falls from dislocating patellæ	Slight, each side	Yes, very perceptible	Apparatus, knee-cap with rubber pad	Some relief.
2	„	M.	14	None known	3¼ inches, L. 3 inches, R.	Walked up and down stairs with awkward gait. Fell at times without apparent cause, dislocating patellæ	Considerable, left side	Yes, on each side	Leg brace giving lateral support	Great relief. Left patella remained unreduced.
3	„	F.	35	None known	—	Walked with great difficulty. Least irregularity of pavement caused fall. Difficulty in going up and down stairs	Slight, each side	No	Light lateral leg braces	Great relief.
4	„	F.	13	Fall on orange peel; dislocated left patella, afterwards right	2¾ inches, L. 2⅝ inches, R.	Falls frequently; knee swells; pain	None	Yes	Knee-cap did no good; leg braces gave relief	Lost sight of.
5	„	F.	—	Breaking down adhesions in joint	2⅝ inches, L. 2½ inches, R.	Disability in joint; click on movement	None	No	Not stated	Not stated.
6	„	M.	—	No cause	3 inches	Difficulty in playing tennis; knee soon gave out. Pain after use of knee, and sometimes heat and swelling	None	No	Knee-caps	No relief; refused operative treatment.

Case.	Surgeon.	Sex.	Age.	Cause.	Ligament measured.	Signs and symptoms.	Knock-knee.	Exostosis between condyles.	Treatment.	Result.
7	„	F.	—	No cause	Elongation very marked	Difficulty in going up and down stairs	None	No	Refused treatment	Not stated.
8	Walsham	F.	21	Knee gave way suddenly whilst walking	3 inches, about	Frequent falls. Pain in knees on walking. Frequent dislocations	None	No	Shortening of ligament by transplanting tubercle	Perfect relief.
9	„	F.	17	Slipped on stone?	2½ inches, about	Constant falls. Looseness of the knee. Could not walk without a bandage	None	No	Ditto	Good; still wearing splint.
10	„	F.	21	None known	—	Knee slips out on slightest provocation, such as treading on loose stone	None	No	Had worn a knee-cap with no benefit; expectant treatment at present	Waiting to see ultimate result in two former cases.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 63.)

SOME CASES

OF

FRACTURE OF LONG BONES FROM SLIGHT CAUSES

IN CONNECTION WITH TABES DORSALIS, SYPHILIS, AND PARAPLEGIA.

BY

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IN the discussion on Charcot's Joint Disease at the Clinical Society in 1884–5 but slight reference was made to the occurrence of so-called spontaneous fractures in association with tabes. Attention was concentrated almost entirely on the joints and the articular extremities of the bones, and none of the speakers adduced cases of tabic fractures either occurring alone or in company with the characteristic articular lesions. This is an indication that tabic fractures are rarer even than tabic arthropathies, and this view is supported by an observation made by Mr. Jacobson that Professor Charcot's cases of spontaneous fracture in locomotor ataxy need confirmation in this country.[1] In a paper illustrating the advantage of rest in cases of locomotor ataxy published in the 'American

[1] Art. "Fractures," 'System of Surgery,' vol. i, p. 405. Reference to Charcot, "Sur quelques arthropathies qui paraissent dépendre d'une lésion du cerveau ou de la moelle épinière," 'Arch. de Phys.,' t. i, p. 161, and 'Diseases of the Nervous System,' Syd. Soc. trans., ser. ii, p. 305.

Journal of the Medical Sciences'[1] Dr. S. Weir Mitchell mentions four cases of fracture of the lower limbs in patients suffering from this disease.

In one the patient broke his thigh, and soon after getting about broke his leg. These accidents he attributed partly to the awkwardness of the patients and the habitual abruptness of the muscular acts, and partly, as he suspected, though it had not been proved, to an impairment of the nutrition of the bones. More recently Dr. Buzzard has placed on record some cases of fracture in association with tabes dorsalis, and has dwelt upon the importance and frequency of the gastric symptoms of the diseases of the nervous system.[2] As it is only by a comparison of observations collected from different quarters that sound conclusions can be drawn, I offer the following additional cases to the Society, accompanied with an expression of regret that they are not all so full or complete as I should have desired to make them.

CASE 1.—*Fracture of right humerus; history of syphilis and locomotor ataxy.*—John M—, æt. 51, labourer, came into the London Hospital under me on the 22nd of January, 1876. Five days before, whilst pulling on his boot, he heard a crack, and found himself unable to move his right arm. On admission it was found that there was a simple fracture of the surgical neck of the right humerus, with extensive swelling of the soft parts over the shoulder. The patient had partial paralysis of both legs, and slight numbness and anæsthesia uniformly distributed over both arms. The pupils were contracted. He was unable to contain his urine. Four years previously he had noticed that his gait in walking was becoming unsteady. He gradually got worse, had pains in his stomach and diplopia, and his powers of progression became seriously impaired. For these and other symptoms he was twice admitted

[1] 'American Journal of the Medical Sciences,' July, 1873, pp. 113—115.

[2] 'Clinical Lectures on Diseases of the Nervous System,' 1882, pp. 85, 231, 236, 237, 250, 251.

into the hospital under Sir Andrew Clark. He had been a free drinker, and there was as definite a history of syphilis as one generally anticipates obtaining from hospital patients with short memories and slender powers of observation. Twenty-six years before he had a slight venereal discharge from his urethra with a hard lump in his groin. He had attended as an out-patient at St. Bartholomew's Hospital for rash over the forehead. His wife had had several miscarriages, but no children born dead. The fracture was treated in the way which I have adopted for years and found useful in cases of fracture of the surgical neck of the humerus, where the tendency of dressers is to apply splints to the lower fragment only, viz. by the application of a weight to the humerus, placed usually, though not invariably, at right angles to the trunk. In this instance a 5-lb. weight was used. A great deal of callus was thrown out, and the fracture united well. If I rightly remember, iodide of potassium was given during the treatment in deference to the suspicious but perhaps inconclusive syphilitic history; and it ought to be added that there had been no sign of a gumma affecting either the humerus or any other bone in the body.

The foregoing case will doubtless be remembered by Sir Andrew Clark and by Mr. R. W. Parker, then Surgical Registrar, who evinced much interest in its ætiology and progress.

CASE 2.—*Ununited extra-capsular fracture of the neck of the femur, with symptoms of tabes in an early stage.*—Alfred A—, æt. 42, a Norwegian or Swedish sailor, was admitted into the London Hospital from the receiving room by Mr. Lipscombe, my house surgeon, on the 3rd of March, 1884. He stated that three months previously, whilst he was carrying a heavy weight on board ship, he twisted round, and suddenly felt something give way in his right hip. He remained in bed for six days after the accident. Then he got up and used crutches, and he had not been able to walk since without their aid. On exa-

mination the right leg measured 31½ inches, and the left 34. Although he had been moving about on crutches, there was very little thickening about the parts or evidence of callus. The limb could be readily drawn down, and crepitus obtained. Under ether extension was made, and a long splint with 15 lbs. weight applied, the shortening being soon reduced to about an inch. At the end of six weeks the patient complained of great pain in his right leg. No cause was discoverable, but it was thought that the extension might have given rise to it, and accordingly the weights were removed. There was then no attempt at union. On the 28th of April, five months after the accident and nearly two since his admission, the patient was discharged at his own request, as he wished to return to his own country. There was still no union. The right hip was fuller than the left, and on making traction and drawing down the leg, instead of crepitus, only a slightly rough sensation was obtainable. The patient stated that he had suffered from rheumatism for four years before the accident, but was not lame during that time. The notes also said that he had had syphilis six years previously, but I could not gather the details which would establish the verity of the affection. The other tabic symptoms noticed in the hospital were absence of knee-jerks, defective sensibility in the lower limbs, and sharp pains in both legs. The pupils acted to light, the eyesight was good, and accommodation normal. As the patient was a foreigner and understood English sparingly, it was not easy to obtain a very satisfactory history.

It will be observed that the extent of the shortening, 2½ inches, scarcely admits of any other conclusion than that the fracture was what is called "extra-capsular." The occurrence of an extra-capsular fracture at forty-two years of age from turning round suddenly is, to say the least, unusual; and equally unusual is it to find no attempt at union and absence of callus, especially under the conditions in which the leg was placed, a week after the accident. All these circumstances point to a constitutional cause,

such as locomotor ataxy, for the fracture in the first instance, and the absence of union and of callus in the second. The precursory symptoms of tabes, with the exception of the eye affection, were present.

CASE 3. *Ununited fracture of the neck of the left femur with definite history of tabes dorsalis.*—William B—, æt. 59, ship joiner, was admitted into the London Hospital on Tuesday, 6th of March, 1884, for a double fracture of the right fibula. On the previous Sunday, being intoxicated and quarrelsome, he had engaged in a row with a fellow-lodger, and was promptly knocked down and trampled on. One of the fractures was situated close above the external malleolus, the other a little higher. He also had extensive bruising on the inner side of the right leg and round the left eye.

Eighteen months before, the patient had been in the hospital under Mr. Hutchinson for left hip-joint disease. Five weeks previously to the first admission he had fallen on his left side, but he walked home without much inconvenience. A few hours later, whilst walking out, he was suddenly seized with violent shooting pains in the left hip-joint, and was unable either to walk or stand. He became an out-patient under Mr. Tay, walking with assistance to the hospital. Whilst in attendance as an out-patient swelling of the hip-joint appeared, and he was taken in under Mr. Hutchinson for "pain and lameness of five weeks' duration." The notes state that there was some swelling in Scarpa's triangle without thickening or shortening of the limb. He remained in the hospital nearly a month, when he was discharged, walking with more ease. He then departed to the hospital in Queen Square, where he was treated for tabes dorsalis, of which he appears to have had the usual symptoms. When he came under my care he was suffering from incontinence of urine, with stricture of the urethra and prostatic enlargement. The urine at first was normal. His extreme restlessness made it very difficult to treat the fracture of the fibula satis-

factorily. Unfavorable symptoms soon manifested themselves—cystitis, fever, bedsores over the sacrum and other parts in contact with the bed, and he died exhausted six weeks after admission. At the post mortem, cystitis and suppurative nephritis were found. The fractures of the fibula had united. The left femur presented only the root of the neck, the remainder having been removed apparently by friction on the dorsum ilii et ischii, on which a new articulation had been formed. In the cotyloid cavity was the remaining portion of the head of the femur exactly filling its cavity, and attached to its floor by the round ligament. Apparently the portions of the neck belonging to both fragments had been worn down by the play of the shaft of the femur.

The specimen is now in the museum of the London Hospital Medical College, and bears date April 16th, 1883. It is entered as os innominatum and head of femur, tabetic arthropathy. In a letter to me dated December 19th, 1884, Dr. Turner says, "I took the specimen of hip-joint to the Clinical Society on the first night of the debate, and had then some thought of making some observations regarding it. It does not, however, show the appearances of Charcot's joint very characteristically. It might be of interest in connection with other cases as presenting changes modified by ataxy."

CASE 4.—*Fracture of right tibia and fibula following disease of the right knee-joint, with definite history and symptoms of locomotor ataxy.*—Maria H—, æt. 55, was admitted into the London Hospital on July 6th, 1883. She had been an out-patient under Mr. Treves for an affection of the right knee-joint diagnosed as Charcot's joint disease, and had been treated for a long time for locomotor ataxy. She suffered from severe lightning pains, and she "walked funny." Her pupils did not contract or dilate either to light or for accommodation. Her right knee-joint was much increased in size. The patella grated freely upon the femur. There was not

much pain in the joint itself, swelling and weakness being the chief symptoms. No other joints were affected. She had been taking Tinct. Ferri Perchlor. with marked relief. There was no history of syphilis, and the family history was good.

The patient was leaving the hospital on July 6th, when her leg gave way under her. She fell and was brought into the building. The right tibia and fibula had given way about three inches below the knee-joint. The leg was swollen and œdematous. It was put up in splints. Rest in bed reduced the size of the knee-joint considerably, but union was slow, and the œdema of the leg had not altogether subsided on the 10th of October. On 31st October, nearly four months after the fracture, the splints were abandoned altogether. There was fair union, but some œdema remained. The patient was ordered a knee-cap and bandage, and was allowed to go home.

CASE 5.—*Extra-capsular fracture of the upper end of the right femur in a patient with symptoms of tabes.*—Maria G—, æt. 46, married, of 58, Cottage Street, High Street, Poplar, a stout, heavy woman, was admitted into the London Hospital under my care on December 29th, 1884. She was carrying a wash-tube half full of water, and twisted her right foot inwards. She felt something give way in her right hip, fell on her left side, and could not get up from the ground. When brought to the hospital she was found to have sustained an extra-capsular fracture of the neck of the right femur. Crepitus was detected, and the fracture was diagnosed to be about the level of the lower border of the great trochanter. The right leg was one inch and an eighth shorter than the left. A long splint was put on and extension applied. Soon after the setting of the fracture a lump was detected about the junction of the upper and middle third of the femur, and this was supposed to be the proximal fragment of the fractured shaft. No signs of this could be felt before the patient was put up in the long splint. On January 14th

an interval was felt between the two ends of the bone, as if the distal end of the proximal fragment was anterior to or riding over the proximal end of the distal fragment. The displacement was rectified by placing a cushion under the lower fragment, and raising it to a level with the upper. On the 20th measurement showed that the amount of shortening had been reduced to three quarters of an inch. On February 9th the splint was temporarily removed and the limb placed between sand-bags. A large amount of callus had been thrown out, and the patient was able to raise the limb from the bed. On the 15th the limb was put up in a Bryant's splint as the union was not firm, and on the 20th the upper and front part of the thigh was painted with iodine liniment, and the application was repeated at intervals of a few days. On March 14th there was only one half-inch shortening. Bryant's splint was still being used, and apparently under the influence of the iodine liniment the callus was disappearing. On the 10th April the splint was removed. On the 15th the patient got up for a few hours, and on the 20th she was allowed to go home. A bandage could not be fixed, but she was provided with crutches for use when she could get about again. On returning home she went to bed, and after some time she was shifted to a chair, and it was three months before she was able to get about with crutches. One day in December she was poking the fire when her husband spoke to her, and she turned round from left to right. She heard a crack, and thought she had re-fractured her thigh-bone. She was taken to the hospital and admitted under Mr. Couper. Shortening to the extent of an inch and three quarters, eversion, and much thickening perceptible to the eye, and looking like a tumour on the upper third of the thigh, were observed, but the surgeon in charge of the case was not satisfied that a re-fracture had occurred. She remained in the hospital a fortnight, and was then sent home, and she remained in bed there for weeks. She almost went out of her mind, was light-headed and

delirious at times, occasioning her husband much alarm. On a Sunday evening three years ago she was coming up the stairs, which are very steep, when one of her crutches slipped, and she felt something crick in her right knee. Since then the right knee has been weak and lax. She notices that when she lies in bed the leg is not straight, but turns inwards below the knee.

I last saw her on the 2nd November, 1892. She was sitting in a chair by her bedside, and her crutches lay on the bed. She never goes out, and can only get about the room a little on her crutches. The right leg is two inches shorter than her left. The callus has all disappeared from the thigh, and as she is very well covered it is extremely difficult to trace the femur. An equal difficulty was experienced when she was first in the hospital. The right knee, tolerably firm when extended, slips about when flexed, and can be bent laterally. She does not sleep well and her appetite is bad. She can see to read a little with the aid of her glasses. The pupils contract for accommodation, but not to light, and she experiences "shocking pains" in her right leg and foot, and sometimes also in the left. She was the subject of lightning pains, and had the Argyll-Robertson symptoms when she was first in the hospital for the fractured femur. There is no definite evidence of syphilis. She has never had any skin eruption or enlargement of glands, but she lost two children in infancy, and had two or three miscarriages.

Her present address is 21, Bow Common Lane, Burdett Road, E. I have only to add that, owing to the thick covering of soft parts, I could not satisfy myself as to the exact line the fracture took in this case, and I believe that it was rather below the trochanter and the neck than through either. She was never examined under an anæsthetic.

By way of contrast to the foregoing cases, I append the particulars of a case of comminuted fracture of the right tibia and fibula in a patient who had been paralysed for fifteen years, and whose limbs were atrophied.

CASE 6.—*Comminuted fracture of right tibia and fibula in an atrophied limb, the effect of paraplegia of fifteen years' duration; good bony union in six weeks.*—Miss Julia McC—, æt. 35, enjoyed perfect health up to the age of twenty years. In the summer of 1869 she was returning home in a steamer from a visit to Margate and was violently sea-sick. Whilst writhing on the floor of the cabin and retching she suddenly lost the power of moving her lower extremities, and she has never walked since. This was followed by wasting of the legs, sloughing over the sacrum with exfoliation of bone, paralysis of the rectum and bladder, great weakness and wasting, nervous debility, and prostration. All the bones of the lower extremities were atrophied. The circulation was feeble. She was accustomed to go out in an invalid chair. One day the butler in lifting her into the chair struck her right foot against the wheel, occasioning a comminuted fracture of the tibia and fibula in the lower third. A great deal of bruising, with the formation of blebs and effusion of bloody serum, accompanied the fracture. The patient was under the care of Mr. Edward Berdoe, of Hackney, who asked me to see her in consultation with him. We put up the leg in splints, and bony union was accomplished in six weeks. From the great prevailing atrophy, extensive comminution, and damaged appearance of the soft parts of the leg, it was feared that bony union might not take place, and that there was some probability of the patient having to lose her leg; but the ecchymosis cleared without breach of surface, and there was no noticeable retardation in the reparative process.

Since this paper was written Dr. Stephen Mackenzie has kindly communicated to me the following case.

CASE 7.—*Transverse myelitis, probably hæmorrhagic, in mid-dorsal region, with secondary degeneration and spastic paraplegia causing "spontaneous" fracture of femur.*—Kate C—, æt. 17, was admitted into the London Hospital on 9th of September, 1892, under Mr. Treves. She had had

no previous illness except measles in childhood, and was living at home in Bromley-by-Bow. For about three months she had been ill and weak; she had lost her colour, and had not menstruated for eight months. On 27th of December, 1890, she got up and breakfasted as usual, and she was sitting at breakfast between 9 and 10 a.m., when she was suddenly seized with violent pain, which went through from her chest to her back. She took chlorodyne and had hot flannels applied to her chest. The pain lasted about twenty minutes, but she sat a little while longer. She had difficulty in getting on to her legs, and when she got up she fell down. She sat again in the chair for two hours, and then had her feet put into mustard and water. It was found that she could not feel the heat, and a doctor was sent for, and not being able to walk or stand, she was carried up to bed. About one week after the seizure the legs used to be painful, but the pain ceased, and she has had no return of it. She was feverish, her temperature five days after the attack being 103°, and she remained feverish about a fortnight. She lost all power over urination and defæcation from the first. A bedsore over the sacrum formed in four days, and there have been sores on the heels, knees, trochanters, and ears, from lying on them. Starting of the legs commenced early, increased, and became very severe. At first when the legs started they used to go down again, but later they became drawn up and were much stiffer. She had no power over them; they would become drawn up every night, and she would push them down with her hands in the morning and during the day as often as they became drawn up. Sometimes they would be straightened out and stiff.

On September 8th, 1892, when pushing the legs down in the morning, she heard and felt a snap in one of them. The leg seemed to swing round. It ached a little and swelled a great deal. A doctor was sent for and she was advised to come to the hospital, which she did the following day. The right thigh was then enormously swollen with

external bruising, but no laceration of skin. Extension with weights from 10 lbs. to 18 lbs. was used, but the leg used to start and jerk very much in spite of them. Gradually the fracture united with an enormous mass of callus.

At the present time there is absolute loss of voluntary power over the legs. Reflex movements are active. The knee-jerk is absent, and there is no ankle-clonus owing to the spastic condition of the limbs. The muscular nutrition is fairly good. There are spontaneous and uncontrollable movements of the legs and constant tonic contraction. The legs are drawn up unless kept down by weights. For the first two days the legs were very cold, but she did not feel it then; they have not been cold since. The only feeling she has is in the right foot. There is vasomotor paralysis. Contact with the skin produces papules and wheals (tache cérébral). Anæsthesia exists from two inches below the umbilicus; paræsthesia from the third rib to two inches above the umbilicus, with a patch of paræsthesia on the left side in the line of the seventh ribs. Sensation about the third rib is normal. There is complete paralysis of the bladder and rectum. The bedsores over the sacrum, left trochanter, and right knee, the general condition, nutrition, and mental state are good. The fracture of the right femur was in the middle third; it is now firmly united, and surrounded by a mass of firm and apparently dense smooth callus of a more or less globular shape.

Remarks.—1. Of the five cases of fracture of the long bones in connection with tabes dorsalis narrated in this paper, it will be observed that in all the seat of fracture was in the immediate vicinity of one of the joints. One case was a fracture of the surgical neck of the right humerus, three cases were fractures of or near the neck of the femur, two on the right side and one on the left; and the fifth case was a fracture of the right tibia and fibula 3 inches from the knee-joint.

2. All five patients were under 60 years of age, the

respective ages being 42, 46, 51, 55, and 57 at the time of the fracture.

3. The causes of the fractures were slight. The humerus was broken by muscular action in pulling on a boot or by slight twisting of the bone during that process. The tibia and fibula gave way under the patient's weight whilst she was going down steps. Two of the femora were fractured through the patients' twisting round whilst carrying a weight. The third femur gave way after a fall whilst the patient was walking.

4. Union of the fracture in the majority was either unaccomplished, defective, or delayed. In two cases the fractures—both being of the neck of the femur—did not unite at all. In a third fracture of the femur the union was incomplete. The patient with fracture of the tibia and fibula was nearly four months in bed.

5. In two of the cases in which union occurred a large amount of callus was thrown out, as observed by Charcot in the case of his patient A. C—, æt. 57, domestic servant, related in the Lectures published by the New Sydenham Society. In one case of union with abundant callus, this callus ultimately became absorbed. Probably absorption is the ultimate fate of callus in tabic patients, although the bones in the immediate vicinity of a joint affected with tabic arthropathy may occasionally undergo great condensation and hypertrophy.

6. The fractures in connection with locomotor ataxy occur like the joint disease in the prodromic stage and before the onset of true ataxic symptoms. They are as sudden and "spontaneous" as the joint affections themselves, and are due to the atrophy and rarefaction without inflammatory changes which are conspicuous in the articular ends of the bones in the joint disease. Both are directly dependent upon lesions of the nervous system.

7. One or two of the cases had a more or less pronounced history of syphilis, but if they were syphilitic it cannot be affirmed that that disease had any direct influ-

ence upon the bones so as to be an efficient cause of the fractures. Syphilitic fractures are usually due to gumma, and may then occur from very slight causes. A case of fracture of the right humerus from gumma was under my care at the London Hospital a few years ago, but after a long search I have not been able to find the notes of the case.

8. Cases of the kind here narrated have in times gone by either passed unrecognised or possibly have been vaguely included under the head of fragilitas ossium or mollities ossium. Even at the present time it is doubtful whether surgeons are sufficiently alive to the association of fracture from slight causes with locomotor ataxia. Whilst Charcot's ataxic joint disease is as familiar as a household word, ataxic fractures can scarcely be said to have attracted the attention which they deserve.

Postscript.

Mr. J. R. Lunn, medical superintendent of the Marylebone Infirmary, has kindly shown me two cases of fracture from slight causes which have been under his care in the wards. The first was the sequel to an example of Charcot's joint disease, contributed by Mr. Lunn to the collection of cases made by the committee of the Clinical Society in 1887 ('Clin. Soc. Trans.,' vol. xx, p. 320).

CASE 8.—Janet S—, æt. 65, was being wheeled out of the garden, when her leg was knocked slightly against the wall. The left tibia and fibula were found to be fractured near the ankle-joint. The limb was put up in plaster-of-Paris, and the fracture united at the end of eight weeks, some deformity remaining. The joint affected was the left knee-joint.

CASE 9.—*Fracture in a case of spina bifida.*—Ellen M—, æt. 12, was born with a large fluctuating swelling over

the lumbar spine. It was not treated, and gradually became smaller and harder. The patient has never been able to walk or stand, and has never had any control over bladder or rectum. There is complete anæsthesia of both legs and the lower two-thirds of both thighs. Sloughing sores on the buttocks and vulva healed under treatment. For five years she has been an inmate of the hospital. She is very fat, and has large breasts.

In January, 1891, the sister noticed that the left leg was unusually limp after the patient had been carried from chapel by a porter. A fracture of the left femur close to the knee-joint was found. It was put up in plaster-of-Paris and united firmly in fair time. The left knee-joint is very lax and moves unnaturally.

The fracture in this case was evidently dependent on the nerve lesions.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 67.)

A CASE

OF

FRACTURE OF BOTH BONES OF THE LEG,

OCCASIONED BY A VERY SLIGHT CAUSE, IN A WOMAN THE SUBJECT OF TABES DORSALIS.

BY

J. W. HULKE.

Received February 28th—Read February 28th, 1893.

E. Q—, æt. 34, a stout, well-grown, good-looking brunette, was received into the Middlesex Hospital on 18th April, 1882.

She related that ten days previously she had broken her leg whilst in the act of hopping on this leg across her room; at the moment of the accident she experienced a sudden sharp pain in the leg, felt a snap, and then fell to the ground. Her health, she said, had never been so good as might be supposed from her "good looks." She had been thirteen years married, and had had one child only, two years after marriage.

About the year 1874 her right foot began to trouble her, it seemed swollen, and a corn, as she supposed it, came in the sole. Later this corn became a "perforating ulcer" in the inner side of the foot. For these troubles

she went into St. George's Hospital, where she underwent several operations, respecting which its Surgical Registrar courteously supplied the following particulars:—"1876, marked anæsthesia of all nerves of the (right) foot; ulceration in sole; great pain. First metatarsal removed, very much thickened. (Later) Syme's amputation for pain and anæsthesia. 1878, nerve-stretching. (Subsequently) excision of 1¼ inches of the sciatic nerve. Functions never altered. 1879, 10th July, re-amputation 2 inches above the stump. 10th October, (leg) removed through the knee."

In the latter part of 1879 she began to experience shooting pains in the left leg.

The condition of her lower limbs at the time she was admitted into the Middlesex Hospital (1882) is recorded in the following note:—The seat of the fracture (in the left leg), sustained ten days since, is marked by a slight angular bend and a lateral displacement at the junction of the middle with the upper third of the tibia. The direction of the fracture is nearly transverse. The surrounding parts are very swollen, and the integument is discoloured by an exceptionally large ecchymosis which extends upwards nearly to the trunk.

The integument of the right thigh, groin, and buttock are completely anæsthetic, with the exception of the following small areas, where tactile and thermal sensibility are much lowered but not quite lost: viz. (1) a small patch at the upper and inner part of the thigh and of the outer surface of the labium majus, continuous with it, innervated by the inferior pudendal branch of the small sciatic; (2) an inguino-pubic area continuous with the upper part of the labium majus, receiving branches of the ilio-hypogastric and ilio-inguinal nerves; and (3) a narrow area in the front and upper part of the thigh, supplied by the crural branch of the genito-crural.

(The innervation of the left lower limb was not at this time investigated.)

The fractured limb was placed in a MacIntyre's splint

and swung. She suffered more frequent and severe pain in the injured limb than is usual at such an interval after the date of fracture. The 23rd of April was marked by an attack of vomiting, unprovoked by any error of diet, which lasted through two days, and ceased after the exhibition of bismuth and hydrocyanic acid.

On May 19th the plantar and dorsal surfaces of the toes were discovered to be anæsthetic, the sensibility of the leg and thigh not being appreciably altered. The insensibility of the surface of the right thigh to tactile and thermal stimuli was unchanged.

June 7th.—Another attack of vomiting, also headache. In the right thigh the area supplied by the crural branch of the genito-crural nerve is now quite anæsthetic.

13th.—Splint removed; the fracture appears firmly united.

On June 25th she returned home. Next day she re-entered the hospital, the apparently solidly united fracture having given way at 10 o'clock that morning, whilst she stood for a moment partially supporting herself as she was being lifted out of a Bath chair. The splint was re-applied. On July 23rd it was replaced by a gypsum bandage, wearing which she left the hospital on August 3rd.

On July 11th the sensibility of the surface of the left leg and thigh was found distinctly lowered, except in the area supplied by the inner branches of the anterior crural nerve. At the date of her final discharge from the hospital anæsthesia of this area had become complete. The circumstances of the fracture prevented electric examination of the muscles of this limb. The further history is, unfortunately, unknown.

It should be added that a careful inquiry into the patient's personal history for evidences of syphilis completely failed.

The circumstances of this case distinctly place it in the class which form the subject of Mr. Rivington's paper announced to be read on the 28th inst.

The pains in the lower limbs, with numbness and associated "perforating ulcer," and also "crises gastriques," clearly indicated the *dorsal tabetic* nature of the nerve disorder. The relatively early age of the patient, eight years less than that of Mr. Rivington's five patients (see *ante*, page 174), the comparatively advanced phase of the nerve disorder when the fracture occurred, and the imperfectness of the union of the fracture leading to refracture by a yet slighter cause than that which produced the original fracture, sixty-four days after the occurrence of this, are points in this case which appear noteworthy.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 67.)

CASE

OF

FIBROUS POLYPUS OF THE PROSTATIC PORTION OF THE URETHRA,

ASSOCIATED WITH PROFUSE HÆMATURIA AND PROSTATIC ENLARGEMENT.

REMOVAL OF THE GROWTH AND RECOVERY.

BY

THOMAS BRYANT.

Received December 28th, 1892—Read March 14th, 1893.

On June 23rd, 1891, I was asked to see Mr. W. P—, a gentleman æt. 63, for an attack of profuse hæmaturia, which was by far the most serious of many similar attacks he had experienced during the past six years.

When I saw him he was feeble and blanched from loss of blood, and his bladder was much distended. On passing a metal catheter with the patient anæsthetised, an obstruction was felt about the neck of the bladder; and with the finger in the rectum the prostate gland was found to be enlarged,—indeed, it seemed as if the gland almost filled the pelvis. It was, moreover, tender and

spongy to the touch. The contents of the distended bladder were blood and ammoniacal urine mixed with ropy mucus. No stone was felt, but the enormous prostate prevented a complete and satisfactory examination of the bladder being made.

On going into the history of the case, I learnt that my patient when young had been a healthy and hard-working man until the age of twenty-three, when he had a gonorrhœa, from which time he had never been able to retain his urine for many hours. He married when he was forty-eight years of age (fifteen years before I saw him), a lady much younger than himself, and he admitted that in a sexual way he had been somewhat free. At times he had suffered from great nerve depression. About seven years previously his inability to retain his urine became more marked, and he micturated about every two hours, at times having to wait some minutes before the stream would flow, and to strain to pass it. Six years ago he had his first attack of hæmaturia, the blood passing from his penis independently of micturition, although when the urine was passed it was mixed with blood. His bladder at that time was examined, but no stone or disease was detected in it. His prostate was, however, then reported to have been much enlarged. Whenever a sound was passed, at this time, for diagnostic purposes, urethral bleeding was produced, when the blood passed away from his penis in clots, unmixed with urine, and the bleeding usually lasted for three days, when it stopped.

Attacks of hæmaturia came on about this time every three or four months, and no causes for them could be assigned; indeed, it may be said that, for the last four years, the attacks had gradually become more frequent, and the intervals between them were rarely longer than two months, and at each attack more blood was lost. Cystitis had complicated the case for the last two years. The patient had been treated by many men, and had taken much medicine, but all to no purpose.

The diagnosis of the case being difficult and obscure, I advised that an exploratory perinæal operation should be made, and I did so under the impression that the cause of the hæmaturia was more prostatic than vesical, and to this the patient consented.

On June 30th the following operation, consequently, was performed. With the patient anæsthetised and placed in the position for lateral lithotomy, a grooved straight staff was passed into the bladder and there held. A median incision was next made in the perinæum, and the urethra opened in its membranous portion as far back as the apex of the prostate, when a Wheelhouse's gorget was run into the bladder.

The bladder was then carefully searched with a sound and finger, and found to be rough and rugous, but free from stone or growth. The prostate gland was of enormous dimensions, and particularly laterally, but the vesical lobe was not sufficiently enlarged to cause obstruction.

On carefully feeling with my left index finger along the surface of the prostatic urethra I detected in a hollow of the floor of the prostate near its apex a polypus, the size of a large haricot bean, vide Fig. 1, Specimen No. 4423A, Royal College of Surgeons' Museum, attached by a narrow pedicle to the floor of the prostatic urethra and projecting forwards, the whole thing being about 1 inch long. This I at once caught with a pair of dressing forceps and removed with scissors (specimen and drawing shown). A drainage-tube was then fastened in the bladder and the patient removed to bed. A steady although tedious convalescence followed the operation, but in about three months after the operation the patient returned home practically well,—that is, he was able to retain his urine for many hours at a time, and to micturate without pain; the enlargement of the prostate had almost disappeared, and the gland felt normal. He passed a catheter

FIG. 1.

Polypus. Natural size.

daily to empty his bladder, for he had not been able to do so by natural efforts for many years. His general health was good. This gentleman is now, a year and a half after the operation, well in all ways. He micturates freely and without obstruction. He has had no urethral hæmorrhage since he left my care, although on several occasions the urine has been blood-stained.

Mr. Targett, the Pathologist to the Royal College of Surgeons, examined the growth and reported as follows:

Report on Mr. Bryant's Specimen of Urethral Polypus.

The tumour submitted for histological examination is pyriform in outline, with a long and delicate pedicle. Its exact size and shape are shown in the accompanying drawing. The surface of the tumour is uniformly smooth and rounded, and on section its substance appears to be homogeneous in structure. Histologically (Fig. 2) the

FIG. 2.

Microscopical drawing of polypus.

growth is made up of interlacing bundles of unstriped muscular tissue, the fibres of which are cut in various direc-

tions. Numerous large vessels are seen in the substance of the growth, and around some of these small cells are collected. There is no capsule, but the peripheral part of the tumour is denser in structure. No trace of an epithelial covering remains; probably this has become detached by maceration. The pedicle is composed of muscular tissue similar to that of the body of the tumour. The attachment of the polypus to the prostatic urethra suggests that it originated as an outgrowth from the prostate, and that its long pedicle was due to its being forced down the urethral passage by the current of urine. It should be added that nothing resembling the glandular tissue of the prostate has been met with in the polypus.

J. H. Targett.

February 16*th*, 1893.

Remarks.—My excuse for bringing this case before the Fellows of this Society is its extreme rarity, and the interesting series of phenomena which were associated with the presence of the urethral prostatic polypus.

It is difficult to believe that the gonorrhœa from which the patient had suffered when quite a young man had anything to do with the origin of the polypus, although the patient asserted with absolute confidence that an irritable condition of bladder which had followed this local affection, had never disappeared.

Seven years before he came under my notice, or one year before the first attack of hæmaturia appeared, this irritability of bladder had increased, and the patient was then unable to retain his urine for more than two hours; at this time it was also noticed that the patient had at times to wait some minutes before the urine would pass, and that he would strain much to pass it. It seems reasonable, therefore, to infer from these facts that at this period the growth probably existed as a source of obstruction.

A year later hæmaturia occurred, and the blood passed from the penis independently of micturition, this fact

clearly suggesting that the blood came from the urethral passage and not from the bladder; and as the bleeding was very profuse, even to threaten life, it must have arisen from the prostate, although it should not be forgotten that the bleeding from the urethra which always took place on the introduction of a catheter may have come from the polypus itself. At this time, however, it was noticed by the surgeons who had been consulted in this case that the prostate gland was enlarged. As years passed the enlargement of the prostate doubtless steadily progressed, and this enlargement, it seems fair to infer, was in a secondary way brought about by the urethral obstruction. For the obstruction excited local irritation, and gave rise to straining in the act of micturition; this in its turn induced engorgement of the prostatic vessels and the spongy enlargement of the prostate gland which existed when the case first came under my notice; and I do not think it can be doubted that this spongy condition of the prostate and the repeated attacks of hæmaturia which steadily increased in frequency and severity during the presence of the urethral obstruction, and which so rapidly disappeared after its removal, were entirely due to the local irritation of the polypus and the straining on micturition which was called forth by the obstruction. What was done, therefore, in the way of treatment was a necessity, and the result obtained fully justified the means.

I should, however, in conclusion like to point out that had a supra-pubic operation been performed here instead of a perinæal, failure must have ensued, for neither the seat of the disease would have been explored nor its true nature been revealed.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 74.)

ON

RESECTION OF THE INTESTINE AND IMMEDIATE SUTURE

IN THE TREATMENT OF GANGRENOUS HERNIA.

BY

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THE treatment of gangrene of the intestine resulting from acute or prolonged strangulation in hernia, is a subject which is still fraught with interest to every surgeon, a subject upon which surgical opinion is still strongly divided, and a subject in which we are again reminded that finality in surgery has not yet been reached. Therefore, in bringing before your notice the details of a case which presents several features of unusual interest, I shall ask your permission to use it as a text upon which to hang some observations upon the subject of the treatment of gangrenous hernia in general, and to use it as an illustration of what I believe I have sufficient material to

prove, that in dealing with gangrene of the intestine in a hernial sac, the more radical operation, the more ideal procedure of immediate restoration of the intestinal canal to its normal state, is at the same time, in the large majority of cases, the best for the patient, both as regards immediate safety and as regards the ultimate results. The notes of the case are as follows:

Miss E—, an unmarried woman æt. 30, was admitted to the Adelaide Hospital on the 22nd September, 1891. Of her family history there is little to record, except that her father died of apoplexy when over eighty years of age, and her mother of cancer of the stomach at the age of forty. Her brothers and sisters are quite healthy. She had never had any serious illness herself until the present trouble began. About three months previous to her admission to hospital she for the first time noticed a small swelling at the umbilicus, which came on after lifting a heavy weight. She could easily push back this swelling into her abdomen without the least trouble or pain. She consulted a lady friend about it, who gave her an abdominal belt, which she had worn for a month before I saw her. During these three months she had suffered much from headache, and complained of a good deal of tenderness over the abdomen, which she found was enlarged.

At 4 a.m. on the morning of the 22nd of September, 1891, she awoke suddenly with a violent pain in the abdomen, and putting her hand down she discovered a large hard swelling at the umbilicus, which she tried in vain to reduce. Vomiting soon set in and was frequent. Dr. Jones of Rathmines saw her about 11 a.m.; he, at once recognising the urgency of her case, sent her into the Adelaide Hospital, where she arrived at 2.30 p.m. I saw her at 5 o'clock, and found her in the following condition. She was a fairly well-nourished, healthy-looking woman, but she appeared to be in a very excited state. Her face was very flushed, and wore an anxious expression. She seemed to be in great pain, which she referred to the

abdomen. Her tongue was furred. She did not appear to be at all collapsed, her pulse was full, regular, and non-compressible. The temperature was normal. Her breathing was rather shallow, and she complained of its being difficult. She stated that she had had an action of the bowels the previous morning, but none since then. The urine was acid, sp. gr. 1030; urates were abundant, but there was no albumen or sugar. On examination I found that the abdomen was uniformly enlarged, and sufficiently tense to prevent any examination of the contained organs. On percussion the flanks gave a dull note; the rest of the abdomen was tympanitic. At the umbilicus there was a tumour about the size of an orange. It consisted of two parts; the upper one small, with a raised rim, depressed in the centre, immediately corresponding to the umbilicus. It was of a reddish colour, evidently acutely inflamed. The lower one, larger, and a little to the right side, was globular, extremely tense, and was of a bluish-black colour. It was hard, very tender to the touch, and firmly gripped at the base. A hard, ring-like mass could be felt encircling its base. No impulse on coughing. The abdomen generally could be manipulated gently without pain, except in the immediate neighbourhood of the tumour. The patient was given a sixth of a grain of morphia hypodermically at 4 p.m. and again at 11 p.m. She vomited a few times during the night, but it was not stercoraceous. She slept at intervals.

Next morning, September 23rd, at 10.15 a.m., 30¼ hours after the onset of the symptoms, the patient was placed under anæsthesia by Dr. Piel with his usual skill; and assisted by Mr. Heuston, I proceeded to operate. I made a median incision directly over the tumour, and when the skin and fascia including the sac, which was adherent, were opened, a loop of small intestine was exposed. It was deeply congested, and at one place was quite black. Several constricting bands were successively divided, and the smaller tumour upwards and to the left

was opened. It appeared like a small pouch, and contained a small portion of intestine. The umbilical ring was very thick, almost cartilaginous. When it was divided the circulation gradually returned to the greater portion of the intestine, but the black portion was quite unaffected, and was evidently beginning to be gangrenous. After a short consultation we decided to perform enterectomy,—in the first place because the intestine seemed incapable of recovery; and secondly, because the portion involved belonged to the jejunum, and an artificial anus, if established, would only at best postpone a fatal issue.

Accordingly I proceeded first to enlarge the original opening at the umbilicus by incising the tissue downwards towards the pubes for about three inches. This was done to enable me to draw out the intestine so as to have sufficient room for suturing. When this was done a quantity of semi-gelatinous clear fluid escaped from the wound. It was so abundant that it was impossible to proceed with the operation until the greater portion of it had been removed. I then inserted my hand into the abdomen to ascertain the cause of this unexpected flow, and I discovered within the abdominal cavity a fairly large ovarian tumour, about the size of a fœtus at full term, attached by a very broad pedicle to the broad ligament at the left side. It was a multilocular semi-solid cyst. We determined to leave it alone for the present. We considered that the length of time necessary to resect the intestine, and to suture the divided ends, together with the inherent dangers of such an operation, constituted a sufficient amount of risk in themselves, without exposing the patient to the danger of prolonging the operation and of adding to it the risks of an ovariotomy.

When a sufficient amount of the glairy fluid had been got rid of to allow us to proceed with comfort, the congested loop of intestine was drawn well forward, and then the abdominal cavity was shut off from the field of operation by means of two large flat sponges, placed one on each side of the intestinal loop, and clamped together

with forceps both above and below the loop. As it is essential in resection of the intestine to cut into absolutely healthy tissues, I determined to divide the gut well above and below the whole strangulated portion; 23½ centimetres (9¼ inches) had thus to be sacrificed. No clamp was used to occlude the intestine, but this end was admirably attained by Mr. Heuston, who grasped the intestine both above and below in his fingers, and thus secured the field of operation against the risks and inconveniences of fæcal contamination. The intestine was divided first above and then below with scissors, and a corresponding wedge-shaped piece was cut out of the mesentery, the divided vessels being secured. Then the edges of the mesentery were united by means of a fine catgut continuous suture. Great care was taken to close accurately the triangular spaces on each side where the mesentery divides to enclose the gut. The method employed was this:—A suture was passed through both layers of the mesentery close up to the intestine on one side; it was then passed similarly through both mesenteric layers on the other side, and firmly knotted. This not only obliterated the triangular spaces, but helped to hold the divided ends of the intestine together. The remaining portions of the intestine were united by means of fine silk (Chinese twist), passed according to Gély's plan. This method differs from the ordinary methods chiefly in this, that the sutures are passed transversely to the long axis of the gut, instead of in a direction parallel to it. The suture only penetrates the peritoneal coat, and although continuous it was knotted at every point of emergence. Only the one row of suture was employed; but in addition, at the convexity of the gut, three Lembert sutures were passed.

The parts were then thoroughly cleansed and replaced within the abdominal cavity, where they lay upon the upper portion of the ovarian tumour.

As much as possible of the glairy fluid in the abdominal cavity was then drained off, and the remainder sponged out. The abdominal wound was closed, and a

glass drainage-tube passed down through the upper portion of the wound. The parts were covered with sterilised sal-alembroth dressings. The patient did not appear at all collapsed after the operation, which occupied an hour and a half. She had a good colour, and the radial pulse was excellent. At 1 p.m. she got $\frac{1}{6}$ grain of morphia hypodermically, and was allowed to suck a little ice. The morphia was repeated at midnight, after which she slept well and had a good night. The subsequent history of the case may be epitomised. The morning following the operation the dressings were changed. They were pretty well saturated with discharge; by means of a syringe and india-rubber tubing $8\frac{1}{2}$ ounces of thick yellowish fluid were sucked up through the glass drainage-tube. The next morning the dressings were again saturated, and $4\frac{1}{2}$ ounces were sucked out. On the fourth day, as no fluid could be drawn out of the drainage-tube, it was removed. The wound healed throughout by first intention, even the track of the drainage-tube closed immediately.

During the period of recovery the diet was limited and very carefully apportioned. From the first day she was allowed a teaspoonful of white wine whey every two hours, and occasionally a little brandy and water. On the fifth day she got a teaspoonful of Denayer's peptonised meat. On the sixth day she was allowed four ounces of milk every four hours, and a teaspoonful of the peptonised milk every eighth hour. On October 1st she got tea and toast, and on October 5th, the thirteenth day, she was put on regular meals. During this period nutrition was maintained by means of nutrient enemata and zymine suppositories alternating every fourth hour. On the day following the operation she passed some flatus, and again on the morning of the fourth day. On the fifth day a warm water enema was administered, which returned almost immediately with scarcely any fæcal matter, but the bowels moved again slightly five hours later, the motion being fluid. The bowels acted with-

out assistance once or twice a day from this date onwards.

On the 27th October, five weeks after the performance of the enterectomy, the patient was in such a very satisfactory condition of health and strength that I considered it would be unwise to delay further the removal of the ovarian tumour. My chief fear was that if I delayed any adhesions which had formed as a result of the former operation would become organised and firm, and would materially increase the difficulties and risks of the operation, especially as, from the position in which the sutured gut lay in the abdomen, the adhesions would form between it and the upper portion of the tumour.

Accordingly, the patient being prepared in the usual manner and placed under anæsthesia, an incision was made from the umbilicus to a point an inch above the pubes. As soon as the peritoneum was opened there was a copious discharge of the same thick gelatinous-looking fluid as at the former operation. The tumour at once appeared, a semi-solid multilocular cyst. The largest cyst was drained through a Lawson-Tait's cannula. The opening in the cyst-wall was then enlarged sufficiently to allow the introduction of the hand, so as to break up as many of the smaller cysts within the tumour as was necessary. When sufficiently reduced in size the whole tumour was drawn out of the abdominal cavity. There were numerous soft adhesions between the cyst-wall and the intestines above, which readily yielded to the fingers. The tumour was attached by a broad pedicle to the whole length of the left broad ligament. There was also a strong broad adhesion binding it down to the right side of the pelvis, almost constituting a second pedicle. Both of these were transfixed and ligatured in sections, and the tumour was then cut free. When it was ascertained that hæmostasis was complete the abdominal cavity was well flushed out with warm boracic solution, 4 per cent., and then carefully sponged dry. I then searched for the sutured intestine. It was impossible to detect it with

the eye, but I soon recognised it by touch. The line of union was so perfect that not even a contraction or depression on the surface marked the line of suture, but with the fingers a thickening beneath the smooth surface could be felt. When the piece of intestine had been examined by my colleagues who were assisting me, the abdomen was closed in the usual manner. The subsequent progress of the case need not be detailed further than to say that on the fourteenth day, November 9th, the patient was allowed up on the sofa, and at the end of four weeks from the time of the second operation she left hospital perfectly recovered. I had an opportunity of examining her six months later. The abdomen was perfectly flaccid, soft, and natural. There was no sign of protrusion at the umbilicus. She was in perfect health, except for occasional twinges of abdominal pain, probably due to flatulency.

The case which I have reported above furnishes us with two distinct subjects which merit consideration. The first of these relates to the co-existence of an ovarian tumour and ascites with strangulated hernia; the second raises the question as to the best means of dealing with the intestine in gangrenous hernia.

I will in the first place allude shortly to the first of these considerations. It is a remarkable fact that the question of how far and under what circumstances the presence of ascitic fluid in the abdominal cavity may influence formation of hernia, has received but little attention. Excluding, of course, the occurrence of acute inflammatory ascites, which may supervene as a result of peritoneal irritation consequent on strangulation, medical literature, by its scantiness, declares that the concurrence of ascites and hernia is sufficiently uncommon. The only attempt at dealing with the question with which I am acquainted is contained in a paper by Prof. Jeannel,[1] of Toulouse. In it he relates a case which in many respects is analogous to mine. The patient, an unmarried woman

[1] 'Revue de Chirurgie,' 1890, vol. x, p. 20.

aged twenty-six, came under his care in 1888 on account of an abnormal enlargement of the abdomen. The abdomen was so distended with fluid that an exploratory puncture was made, and 12 litres of a clear lemon-coloured fluid were evacuated, but all the liquid was not removed. It was then found that the cause of the ascites was a tumour in the left iliac fossa. Two days later, whilst straining at stool, an umbilical hernia, which she had had for eighteen months, became suddenly strangulated. Four hours later, taxis having proved unavailing, herniotomy was performed, and the gut liberated. The incision was then enlarged so as to admit of the removal of the tumour, which proved to be a parovarian cyst of the left side. In closing the abdominal wound a radical operation for the cure of the hernia was performed. The patient made a good recovery.

I have epitomised this case because it so closely resembles the one I have just reported; only in Jeannel's case, the strangulation having only existed for four hours, it was possible to relieve the patient by a simple herniotomy, without having to interfere with the intestine itself.

Although in some works on hernia the presence of ascites is mentioned as a predisposing cause of hernia, it is interesting to note how very few cases of this actual condition are reported.

Professor Jeannel has succeeded in collecting only eight cases, two of them reported by himself; and I have only been able to add one[1] to the list in addition to the one case I have observed myself, although I have searched the 'Index Medicus' and many other sources for cases of this kind, but without success.

Of these ten cases we find that eight were umbilical, one inguinal, and one crural. The preponderance of umbilical cases is what one would suspect. In fact, it would seem that fluid in the abdomen would tend to prevent inguinal or crural herniæ. In nearly all the

[1] Case 118 in tables.

usual positions of the body the weight of the fluid, gravitating downwards, would displace the intestines, and keep them away from the femoral and inguinal rings. The tendency of the intestines to float on the fluid, while saving them from the dangerous proximity of these lower rings, would, on the other hand, approximate them to the umbilicus. Their movement in this direction is limited only by the length of the mesentery; and should this be normal we can understand how a very abundant ascites, enormously distending the abdominal wall, might prevent a hernial accident at the umbilical ring which a more limited amount of fluid would promote.

In fact, in the cases quoted by Jeannel we find that the amount of fluid was not excessive. In some it had never been in great quantity, whilst in others strangulation of the hernia ensued after the abdomen had been partly evacuated. Hence, as Jeannel points out, "it seems that in ascites, strangulation of an umbilical hernia is specially to be dreaded when the ascitic fluid is but scanty, or when, having been evacuated, the abdominal wall is but slightly distended."

I will now pass on to the consideration of the larger, and perhaps the more vital question, namely, the best method of dealing with gangrenous intestine in a hernial sac.

Perhaps the most commonly employed method up to recent times is that with which the name of Sir William Lawrence is identified: it consists in "a free incision through the mortified part, in order to unload the distended intestinal canal; or, if the gut should have already given way, to divide freely the integument and sac, and to leave the subsequent process of cure entirely to nature."[1]

The principle which underlies this method of treatment has been very generally adopted, but many modifications have from time to time been introduced, which have been received with more or less favour. Thus some advise that the gangrenous loop should be left *in situ* unopened, as

[1] 'A Treatise on Ruptures,' 5th edition, 1868, p. 364.

this gives better opportunity for plastic exudation to seal the parts, and so prevents the poisonous materials entering the abdominal cavity. As far as relieving the obstruction is concerned, it matters little whether the intestine be opened or not, as if left to itself the gangrenous portion will crumble away within thirty-six hours. Many operators recommend that the stricture should be divided in order to give free vent to the contents of the bowel above the seat of strangulation, and to allow the unhealthy portion of intestine to be well drawn out. Others, again, as strenuously oppose this proceedure. "My own very strong opinion is," says Mitchell Banks,[1] "that the stricture should be left alone. . . . I maintain that any dividing of the stricture, involving as it does the risk of opening a passage into the peritoneal cavity from the putrid external wound, is apt to provide a channel for the subsequent leakage of septic materials into that cavity. . . . Slit up the gut, take off all tension, and let its juices run out, and there will soon be plenty of room for flatus and fluid fæces to escape."

On the other hand, Professor Spence said, "I divide the constriction freely, and draw down the gangrenous portion of gut beyond the stricture, and leave it in the wound merely covered by lint soaked in warm water. Some hours afterwards I open the gut and cut off the gangrenous portion, so as to allow the fæces to escape. By that time lymph has been effused around the protruded gut, and the risk of fæculent fluid passing back into the abdomen is very much lessened."

A step further in the treatment of these cases on Lawrence's principle consists in opening the intestine and suturing it to the wound. As recently as 1888 we find this treatment recommended by Mr. Jordan Lloyd,[2] who successfully treated a lady aged forty-five by this method. I shall have occasion to refer to this case again further on.

These various methods of dealing with gangrenous

[1] 'Proceedings of the Medical Society of London,' vol. viii, 1885, p. 289.

[2] 'Birmingham Medical Review,' vol. xxiv, 1888, p. 158.

herniæ agree in this, that they all aim at establishing an artificial anus, and they leave it to nature to perform. It was felt, however, that in many cases surgical art could do better than nature, and this led to perhaps the most important modification of Lawrence's method, namely, the immediate formation of an artificial anus by the surgeon's skill. Shortly, it consists in at once excising the damaged piece of intestine and suturing the healthy ends of the gut to the incision in the skin. This is the method which is perhaps most frequently adopted at the present day. But is it the best?

There is one great disadvantage under which all operations for the establishment of an artificial anus for the cure of a gangrenous hernia, labour, namely, that a second operation for the cure of the artificial anus becomes a matter of necessity. The irritation and excoriation of the skin around the anus, caused by the continual passage over it of the fluid contents of the small intestine, make life frequently unendurable; whilst a much more serious danger not uncommonly presents itself in the rapid exhaustion of the patient from mal-nutrition caused by shutting off a long piece of intestine from the food channel, and consequently leaving a diminished area in the intestinal tract for absorption.

The secondary operation may follow the lines laid down by Dupuytren, and the continuity of the canal may be restored by the use of an enterotome. This method finds favour with many, because this secondary operation is apparently a comparatively safe procedure. In the forty-one cases reported by Dupuytren[1] in his 'Leçons orales' but three deaths occurred, or 7·3 per cent. On the other hand, however, it is not by any means always successful. It is not uncommon to find in the record of cases so treated, in recent years, the statement made that the patient "continues to have a preternatural anus." In Dupuytren's forty-one cases the anus persisted nine times, or in other words the operation failed in 21·9 per cent.

[1] 'Leçons orales,' vol. ii, 1832, p. 286.

Instead of resorting to the enterotome, secondary resection and suture may be adopted. In favour of this method we may point out firstly, that it seldom fails to effect its object. Thus in the list of cases collected by Mr. Makins,[1] the operation failed only three times among 39 cases, that is a percentage of 7·7 per cent. That is, the treatment by the enterotome for the cure of artificial anus fails about three times as frequently as when secondary incision and suture are employed. Secondly, when a cure is obtained, it is brought about in a much shorter space of time by means of resection. Three to four weeks is generally sufficient for cure; whilst with the enterotome the operation has frequently to be repeated, and the cure when effected takes months. Thus in Mr. Jordan Lloyd's case, already referred to, it was nearly twelve months before the fistulous opening was finally closed.

On the other hand, the cure of artificial anus by resection has a much higher death-rate. In Mr. Makins' table, containing 39 cases, death occurred fifteen times, which gives a percentage of 38·4. Among 29 cases collected by Bouilly,[2] the operation failed only once, but death occurred in 11 cases, a percentage of 37·7. These statistics so nearly agree, that I think we may fairly assume that the death-rate of enterectomy and suture for the cure of artificial anus is about 38 per cent.

In estimating the relative value of the different methods of treatment, and in endeavouring to arrive at truthful deductions, it is perfectly fair to draw comparisons between these two methods of dealing with an artificial anus resulting from gangrene of the intestine in hernia; because they both start from the same point, and they both seek to arrive at the same end. The conclusion which we may, therefore, draw between these two operations is, that for the cure of artificial anus resection of the intestine and suture is far more fatal than Dupuytren's

[1] 'St. Thomas's Hospital Reports,' vol. xiii, 1883.

[2] 'Revue de Chirurgie,' vol. iii, 1883, p. 549.

method, but on the other hand it yields more certain and quicker results.

In competition with all these methods of treating gangrenous hernia, is the plan which has been employed, if I may so say, sporadically for a great number of years, but which has only in recent years received consideration as an operation which deserves to be employed more generally than has hitherto been the rule, and that it should not be reserved merely for exceptional and rare cases.

It is acknowledged on all hands that resection and immediate suture in gangrene of the bowel in hernia is, in the words of Helferich, "a perfectly ideal operation."[1] When successful, the patient is well and restored to his normal condition in from three to four weeks. He has no secondary operation to undergo. This operation, then, should obviously hold the first place, if we could show that the risks attending it were not greater than the risks of forming an artificial anus and subsequently curing it. The question, then, as to which operative procedure should, as a general line of practice, be adopted, resolves itself mainly into a question of statistics, and I hope to show that in this matter statistics speak with no uncertain sound.

The opinion generally held, certainly in these countries, is that the mortality following resection and immediate suture is sufficiently greater than the mortality following the older operation to outweigh the obvious advantages of the former method, and therefore the rule in general force is to restrict the operation of resection and immediate suture to the exceptionally favorable and more promising cases, and in the large majority of gangrenous herniæ to establish an artificial anus.

I shall presently discuss the soundness of this view, but before doing so I should like to point out the extremely faulty premises upon which this conclusion is based. I have already pointed out that it is perfectly legitimate to compare the results of secondary suture for

[1] 'Archiv für klinische Chirurgie,' Band xli, 1890, p. 337.

the cure of artificial anus with the employment of Dupuytren's enterotome, and to draw conclusions from the comparison as to the relative merits of the two procedures. But it is illegitimate to compare the results of primary resection and suture for a gangrenous hernia with secondary resection and suture for artificial anus, the result of a gangrenous hernia, and because we find the mortality of the former is higher than the mortality of the latter, to at once jump to the conclusion that the establishment of an artificial anus is the safer method of treating a patient whose hernia has become strangulated and gangrenous.

If we are to compare the mortality of the two procedures, we must place on one side the percentage of deaths following the primary operation of resection and suture. On the other side we must place first the mortality which follows on the operation for forming an artificial anus; to this we must add the mortality of the secondary operation. When this is done we shall be in a position to compare the two operations. But this is not the basis upon which the conclusion which is so generally accepted rests. It would seem as if many writers on this subject, and men whose views carry weight, look upon this matter as if, in dealing with these cases, the surgeon had three alternatives from which to choose,—whether he would form an artificial anus, whether he would resect and suture at once, or whether he would resect and later on suture or use the enterotome. Whereas it is obvious that he has practically only two, to do the whole operation at once, or to divide it into the two parts; and the mortality of the first must be compared to the combined mortality of the operation *à deux temps*. I lay particular emphasis upon this point because I find in English literature it is so frequently ignored. For instance, one of the most frequently cited statistics in the English language is quoted from a valuable paper in the 'St. Thomas's Hospital Reports' by Mr. G. H. Makins.[1] In this paper the author, referring to the

[1] Vol. xiii, 1883, p. 193.

table of cases which he himself had collected of gangrenous hernia treated by excision and immediate suture, says, "In the table below are 55 cases with 29 deaths, a percentage of 52·7, against a percentage of 38·4 in cases of resection for artificial anus. . . . . A glance at the statistics serves to show the very great advantage of carrying out the resection of the intestine when it is in a comparatively normal condition, and *hence they favour very decidedly the older method of establishing an artificial anus* in cases where the condition of the gut precludes any idea of its return into the abdominal cavity."

Now this last deduction does not follow as a logical consequence of the statistical premises. It simply assumes that the mortality attending the operation for the formation of an artificial anus is *nil*, or so small that it may be neglected altogether. I will give another illustration. In the 'Edinburgh Medical Journal'[1] Mr. J. M. Cotterill relates a very interesting case of gangrene of the transverse colon in an umbilical hernia, in which he excised 15 inches of the colon and formed an artificial anus. Over five months later he resected 7 inches more and sutured, the patient recovering. Commenting on this case he says: "It has been established chiefly by statistics . . . . . that the best results after resection of intestine are obtained in cases of operation for the cure of artificial anus. . . . In other words, it appears less hazardous to do the operation in two stages rather than complete the operation at the time of the removal of a gangrenous portion."

This is again clearly a case of *non sequitur*, and yet it is based upon such arguments as these that the opinion prevails that resection and immediate suture in gangrenous hernia is more fatal than forming an artificial anus and subsequently curing it. Thus Mr. Mitchell Banks[2] writes: "We have to consider which is the better plan, to procure

[1] 1889, vol. xxxiv, pp. 602—607.

[2] 'Proceedings of the Medical Society of London,' vol. viii, 1885, p. 285.

a patient an artificial anus and cure him of that subsequently, ... or to subject him at once to an operation *more fatal, it is true,* but which in all other respects manifestly has the advantage."

I have given these extracts in order to show how deep rooted is the belief that the immediate plan of treatment is more fatal than the operation divided into two stages. Now let us sift this matter carefully and see what the truth really is.

When I first began examining the statistics of these operations, I was quite unprepared for the extraordinarily high death rate which follows the operation for establishing an artificial anus in cases of gangrenous hernia.

A collection of isolated cases from all sources sometimes gives a false impression as to the mortality of a given operation. It is always open to an objector to say that especially in any novel operative procedure, the successful cases are nearly always published, the fatal cases frequently escape appearing in print. In the present instance we shall fortunately escape this objection. I have made no attempt to tabulate isolated cases of gangrenous hernia treated by the formation of artificial anus, but I can give the results of this method as tested by individual operators in various large hospitals and kliniks, where each operator records, not isolated cases but every case treated by this method under his own observation. Such statistics are of the greatest value and can be relied on.

Thus Korte[1] records 28 cases of gangrenous hernia in the Bethenien Hospital, which were treated by the formation of an artificial anus; 10 died shortly after the operation, either from collapse or peritonitis; of the 18 remaining, 6 died (three from infection of the wound, two from intercurrent diseases, and one from inanition). Death-rate, 16 in 28 cases or 57 per cent.

In the 'Friedrichshain' at Berlin 26 cases of gan-

[1] 'Deutsche med. Wochensch.,' No. 41, 1888.

grenous hernia were treated by artificial anus, of which 20 died, a mortality of 76·9 per cent.

Poulsen[1] records 29 similar cases of which 25 died, a mortality of 86·2 per cent.

Of 15 cases treated by the formation of artificial anus by Reichel in the klinik at Breslau, 12 died, viz. 80 per cent.

Kocher of Bern, who has recently published all his cases of gangrenous hernia, operated seven times by forming an artificial anus. Of these 7 cases only 1 recovered, showing a mortality of 85·7 per cent.[2]

Cohn[3] has recently given the results of all his herniotomy cases, 220 in number, treated between the year 1880 and the summer of 1888; 26 gangrenous cases were treated by the formation of an artificial anus and 21 died, a mortality of 87·7 per cent

Such are the results when the artificial anus is made by the surgeon; but are the results any better when the formation of abnormal anus is left to nature. ?

Ill[4] collected 29 cases treated in this manner in the United States, and in these 25 patients died, giving a mortality of 86·2 per cent. These statistics may be objected to on the ground that they are isolated cases, and do not represent all the cases operated on. But we can refer to statistics on this point which are above suspicion. In a most valuable communication read before the Royal Medical and Chirurgical Society in March, 1891, Mr. C. B. Lockwood has given us the results of all the cases of hernia in which the strangulated gut was ulcerated or gangrenous, and all of which had been treated in St. Bartholomew's Hospital during the past seventeen years by free incision. He found 35 cases so treated, besides 2 in which he had employed the same method himself. Added to these there were 5 operated on in this way in

[1] 'Hospitals Tidenda,' 1889.

[2] 'Deutsche Zeitschrift für Chirurgie,' Band xxxii, 1891, p. 182.

[3] 'Berliner klin. Wochensch.,' 1889, Nos. 20 and 21.

[4] 'Med. Record,' Sept. 22nd, 1882.

1890, making a total of 42 cases. Of these 38 died, showing a mortality of 90·5.

In the accompanying table in which I have arranged these statistics, I find that out of 202 cases treated by the formation of an artificial anus 163 died, giving a mortality of 80·7 per cent.

Table of Cases of Gangrenous Hernia treated by the Formation of an Artificial Anus.

	Cases.	Recovered.	Died.
Korte	28	12	16
Friedrichshain	26	6	20
Poulsen	29	4	25
Reichel	15	3	12
Kocher	7	1	6
Cohn	26	5	21
Ill	29	4	25
Lockwood	42	4	38
Total	202	39	163
		19·3 %	80·7 %

Let us now consider what is the death-rate following the operation of resection and immediate suture in cases of gangrenous hernia.

Owing to the fact that very few operators have had a sufficiently large experience of this method to enable us to draw conclusions from the work done by a single operator or in a single hospital, I have been obliged to depend mainly upon the results as shown by a collection of all the cases I could find recorded in medical literature up to date.

I have succeeded in tabulating 222 cases, of which 104 died, which gives a mortality of 47 per cent. But it may be said that this does not represent the true death-rate, as many fatal cases are unreported. Among the list of operators, however, we find the names of three who have had exceptional experience in this operation. Kocher of Bern, Hagedorn of Magdeburg, and Mikulicz have recently published all their cases of resection of the intestine.

The cases extracted in my tables comprise only those in which resection and *immediate* suture were employed in gangrenous hernia, and in which a cylinder of the whole circumference of the gut was removed. I have thus tabulated 19 cases by Kocher, of which 9 recovered and 10 died, a mortality of 52·6 per cent. Sixteen cases by Hagedorn, of which 7 recovered and 9 died, a death-rate of 56·25 per cent. Twenty-one cases by Mikulicz, of which 14 recovered and 7 died, a death-rate of 33·3 per cent.

Putting all these cases together, as we have done in the case of artificial anus, we find a total of 56 cases by these three operators, of which 30 recovered and 26 died, or a death-rate of 46·43 per cent. This substantially agrees with the percentage of deaths obtained from the larger table of 222 cases.

But it must be acknowledged that the death-rate is materially improved by the brilliant results obtained by Mikulicz; but even if we rely on the results of Kocher and Hagedorn, and accept the mortality following this operation in their hands as the probable death-rate which attends resection and immediate suture, we have a mortality of 54·3 per cent. at most. Compare this with the death-rate attending the formation of artificial anus, which we have shown to be 80·7 per cent., and I think we must agree that it is unnecessary to add to this latter the death-rate of the second operation which is then necessary, either of secondary resection and suture, or of the use of the enterotome, to convince us that primary resection and suture is not only a more ideal operation, and has in every other way the advantage, but as regards risk to life it is immeasurably superior to the method of establishment of an artificial anus, independently altogether of the fact that the latter necessarily involves a second operation later on.

It may be objected, however, that the difference in the death-rate between these two operative procedures depends upon the nature of the cases in which the operations are performed. It is laid down by many writers that resec-

tion and immediate suture should be reserved for the more favorable class of cases, whilst in all the more severe forms an artificial anus should be established.

Bouilly[1] formulates the conditions under which resection and immediate suture may be undertaken.

1st. When the state of the patient is not so bad as to make us fear the length of time required by the operation, and the prolonged administration of anæsthetics.

2nd. When a careful examination allows us to reject the existence of a generalised peritonitis, or of any serious complication.

3rd. When we can assure ourselves at the time of operation that there has been no extravasation of fæcal matter into the abdominal cavity.

4th. If we believe we can with ease draw out the whole of the gangrenous portions of intestine and mesentery and resect in healthy tissue.

5th. If we can re-establish in a solid and efficacious manner the continuity of the intestine without being hampered by too great a difference between the calibre of the two ends.

These conditions, sufficiently rare to meet with in gangrenous hernia, have been accepted as the canons of practice by most operators hitherto. They are well expressed in the words of Coppens[2] of Lille:

1st. "Simple enterotomy, followed by the formation of artificial anus, should be the operation of choice in gangrenous hernia.

2nd. "Intestinal resection, more dangerous, should be reserved for absolutely special cases, and should be considered as an exceptional procedure."

Now I think these canons will have to be revised and considerably modified. It may have been true that resection has been reserved for those cases which promised well, but it is not true generally to-day. For look at the brilliant results obtained by Kocher and

[1] 'Revue de Chirurgie,' vol. iii, 1883.

[2] 'La Clinique,' 3e année, Nos. 10 and 11.

Hagedorn, both of whom have practically abandoned the operation for the formation of artificial anus, and resect and suture in every case, even in the most unpromising. I think their results show that there are a large number of cases in such a condition of collapse or of septicæmic poisoning when they first present themselves, that they are beyond the reach of surgical skill; but if any such cases are to be saved at all, they run a better chance with resection and immediate suture than if an artificial anus be formed. Far be it from me to say that in no case should an artificial anus be established. There are extreme cases where the condition of the patient is such that the smallest amount of surgical interference or of anæsthesia can be badly borne; in such cases we must do what we can—relieve the obstruction,—but we can have but little hope in a successful issue.

Looking at the statistics I think what they mean is this, that the death-rate which follows resection represents fairly accurately the number of cases that will die in gangrenous hernia, whatever method be adopted for their relief; and that the difference between this mortality and the death-rate following on artificial anus indicates the number of cases which might be saved by the former operation. We might then formulate our conclusions thus:

1st. Intestinal resection and immediate suture should be the operation of choice in gangrenous hernia.

2nd. Simple enterotomy, followed by the formation of artificial anus, should be reserved for absolutely special cases, and should be considered as an exceptional procedure.

I am aware that these views are not shared in by many of our most eminent surgical confrères; but I think this is mainly due to the fact that the conclusions to which the statistics point have not been appreciated, and in the absence of full statistics, it is natural to suppose that removing a piece of intestine and sewing the divided ends together at once must necessarily be a more dangerous proceeding, than simply to incise the

intestine and relieve obstruction in the most simple manner possible. If the real state of the case had been known, I do not think Mr. Jordan Lloyd,[1] for instance, in publishing a case in which he formed an artificial anus, and subsequently succeeded in closing it by Dupuytren's method after a treatment which lasted nearly twelve months, would have written:—"I am led to publish this case chiefly on account of the successful result of the treatment, because there is a growing tendency (amongst that school of surgeons who regard operation as 'the all and the everything') to deal with gangrenous intestine *by more radical and more rapid and certainly more risky* methods."

There are several great dangers to be encountered, and, if possible, avoided in cases of gangrenous hernia. The first of these is the great distension of the afferent piece of intestine, which is generally filled with great quantities of fæces and gases. As a result of this distension we may find this portion of the intestine in a paralysed condition, incapable of maintaining the circulation of its contents, even after the obstruction has been relieved and the continuity of the canal restored. We find then that the symptoms of obstruction persist after all our attempts to relieve it. In such cases it is recommended by Julliard to form an artificial anus, and to resect and suture in two or three weeks. But in many of these severer cases the exhaustion of the patient steadily increases when an artificial anus is formed, and hence Riedel proposes, and he is supported in his recommendation by many of the most experienced operators, that after an artificial anus is formed, secondary resection and suture should be carried out at the earliest possible moment. In the investigations I have made for the purposes of this communication, I have come upon many cases in which secondary resection and suture has been done within twenty-four hours, and frequently with gratifying results. Kocher, however, prefers to resect and

[1] 'Birmingham Med. Review,' vol. xxiv, 1888, p. 162.

suture at once. He considers that as it is necessary to draw the loop of intestine well outside the abdominal cavity, it is easy to empty the afferent portion of gut of its contents after it has been divided and shortly before the application of the sutures. Another modification has been proposed. It was originally suggested by Helferich[1] in cases where the exact condition of the intestine was doubtful, and it was not considered wise or safe to return it to the abdominal cavity at once. He advised to leave the suspected gut outside the abdomen, well protected with dressings, after having performed intestinal anastomosis between the efferent and afferent portions of intestine. This suggestion, which has been favorably received, promises to be of use in the class of cases we are considering. Should this exposed loop prove to be gangrenous it can be excised the following day without recourse to anæsthesia, and the ends of the bowel inverted and closed by Lembert sutures. Helferich has tried this method twice, once with complete success, the second case dying in a few hours of collapse.

Another source of danger which may present itself in cases of gangrenous hernia is the severely damaged condition of the walls of the afferent portion of the intestine, which are frequently œdematous and congested, sometimes bordering on gangrene. It is extremely difficult always to estimate with any degree of certainty the exact limits of the injury. We find, in many of the fatal cases reported, that the cause of death was attributed to gangrene spreading upwards in the afferent portion above the seat of suture. On the other hand, we find that recovery has followed when large portions of the intestine have been removed. Thus Ramdohr's case (No. 1) in which he excised 2 feet, Rydygier's case (No. 29) in which 54 cm. were sacrificed, Rushton Parker (No. 89) who cut out 12 inches, Walter (No. 103) who removed 2 feet 4 inches, and lastly Kocher (No. 165), whose patient left hospital perfectly well on the eighteenth

[1] 'Archiv für klin. Chirurgie,' Band xli, 1890, pp. 337—345.

day, after having had 1 m. 60 cm., about 5¼ feet, of intestine removed. All these patients recovered. Thus we see that great danger may result from removing too little, but that within limits the risk is not increased by removing a long piece of the intestine. Therefore, in excising the damaged loop in gangrenous hernia, we should not hesitate to sacrifice all that appears in the slightest degree suspicious. The gut should be divided on both sides through absolutely healthy tissue. To obtain good rapid union and to avoid leakage the sutures must always be passed through healthy intestine.

If there be any doubt about the security of the suture, any risk of leakage, it becomes a matter of importance to diminish as far as possible any pressure of fæces and gases which the intestine may contain. With this object in view Bouilly[1] has recommended a "mixed method." He first resects in the usual way, then he sutures the ends together by Lembert's plan the whole way round, with the exception of a piece about one centimetre in length on the convex border of the intestine, opposite its mesenteric attachment, where he leaves a small opening. The edges of this opening he sutures to the abdominal wound, thus forming a fæcal fistula, which Bouilly maintains will close spontaneously in a short time. Whilst approving of this principle in certain cases of forming a "ventilator," Kocher prefers to make a very small opening in the healthy intestine above the situation of the fully completed suture. The suture-line will thus be less liable to be disturbed by the fæces.

I do not purpose in this communication to discuss the causes of death in gangrenous hernia when treated by resection and immediate suture. Fatal results due to faulty technique are, as experience grows, becoming rarer, and the statistics are to a certain extent from this cause improving, and there can be little doubt that they will in the future bear even higher testimony to the care and skill of the surgeon. But the high mortality attendant

[1] Loc. cit.

on gangrenous hernia is due mainly to two causes which the operator cannot remove, but which medical practitioners generally would do well to lay to heart. They are (1st) the length of time strangulation of the bowel is allowed to go on before surgical assistance is sought, and (2ndly) the injudicious use of taxis. These points are well emphasised in Kocher's cases. The duration of strangulation did not exceed three days in any of the cases which recovered after resection and suture, with one exception, a remarkable case of Littré's hernia, in which the strangulation had lasted for seventeen days. On the other hand, it is remarkable that death occurred twice only in herniæ which had been strangulated for only two days. In both of these cases, however, the patient had been subjected to prolonged and fruitless attempts at reduction by taxis before seeking surgical aid in the hospital.

Thus Bardeleben[1] says in his surgery, "One may assert with justice, that among ten unfortunate results of operation, nine are due to waiting too long and to too violent and forcible attempts at reduction by taxis."

So much has been written of late years as to the various methods of suturing the intestine, and as to the best means of carrying out the different technical details, that it is unnecessary for me to discuss them at length. I need not do more than point out what experience seems to indicate as the best methods to adopt.

In dealing with the mesentery it will be observed that in most of the earlier cases a wedge-shaped piece has been excised. Where portions of the mesentery are gangrenous, this is probably the best plan; but where the mesentery is sound, it is now generally advised that no portion of the mesentery should be sacrificed, as any interference with its vessels tends to increase the risk of gangrene in the portions of the intestine sutured.

Kocher's method is to ligate the mesentery, either singly or piecemeal according to the length of intes-

[1] Vol. iii.

tine to be removed, parallel to the gut. Hagedorn's plan, described by Habs,[1] is to transfix the mesentery about two centimetres from the gut opposite the centre of the portion to be excised, and by passing ligatures through the mesentery to ligate it piecemeal, the last knot being tied at a spot about one centimetre distant from the place where the intestine is to be divided at each side. The object of thus stopping short of the site of division is to protect as far as possible the sutured ends of intestine from becoming gangrenous. The mesentery is then divided close to the intestine through the whole length of the piece to be excised. This is also the method adopted by Mikulicz in all his cases, and has the great advantage of being rapidly performed.

Of all the various methods of suture the one most in favour is unquestionably Lembert's. Some prefer to use an interrupted suture, some a continuous, sometimes both are used in the different rows, but in suturing the serosa Lembert's method is, in the great proportion of cases, adopted. Hagedorn always employs a continuous suture, and, as it seems to me to be a form of suture that will find favour with many surgeons on account of its simplicity and the rapidity with which it can be introduced, I will shortly describe it. The ends of the intestine are cut across obliquely, so that more is taken from the convexity than from the concave or mesenteric side. For suturing the mucosa a strongly-curved needle (half circle) is used ; for the serosa a less-curved needle (three-eighths of a circle). The first or mucous suture begins at that portion of the intestine corresponding to the mesenteric attachment. The needle transfixes, beginning from within outwards, first the mucosa, then the muscularis of one side, then back again through the muscularis and mucosa of the other end of the intestine. The suture is then knotted, but the ends are not cut off. The two ends of the intestine are now sewn together with a continuous

[1] "Bericht über 200 Herniotomien," 'Deutsche Zeitschrift für Chirurgie,' Band xxxii, 1891, p. 357.

stitch until half of the circumference has been gone round, transfixing always in the same order, viz. mucosa, muscularis—muscularis, mucosa. So far the suture has been applied, sewing from the inside. When the centre of the convexity has been reached the order must be reversed, as we must sew from the outside. The needle, therefore, transfixes one side only, about 1—2 millimetres back through the mucosa and muscularis. The continuous suture is now carried on round the other half of the circumference in the reversed order, viz. muscularis, mucosa—mucosa, muscularis, until the mesenteric border is again reached. The suture is now knotted to the end left free at the first knot. This ensures that the knot shall be drawn within the lumen of the gut, or with a blunt instrument it can be pushed in. The blunt probe now pushes in the edges all round, to make certain that mucosa shall lie against mucosa and muscularis against muscularis. The second or serous suture is also continuous. It begins at the mesenteric border, and is carried the whole way round and finally knotted at the mesenteric border. It is applied according to Lembert's plan. This method of suture has been employed in all Hagedorn's cases, and, as will be seen by consulting the column "Remarks" in the table, even in the fatal cases the suture never failed.

The material employed for suturing the intestine seems to be a matter of choice. Many surgeons, among whom is Kocher, prefer silk; catgut has an equally strong clientèle. Hagedorn always employs catgut, for he says that union is so rapid between the serous coats of the two ends, that catgut holds quite long enough, and it is less likely to give rise to subsequent trouble.

A great many new and ingenious methods have lately been described for performing enterorraphy, mainly with the object of preventing a narrowing or constriction of the gut at the site of suture. Among these may be mentioned F. B. Robinson's[1] invagination method; Bowreman

[1] 'Annals of Surgery,' 1891, vol. xiii, p. 81.

Jessett's[1] method, a form of invagination with the assistance of decalcified bone tubes; and lastly, a method of suturing the ends together, giving one end a half twist so as to bring the mesentery of one cut end in apposition with the convex surface of the opposing end, described by Connell of Milwaukee.[2] But ingenious as these methods are, I doubt very much if they are ever likely to supplant the more simple methods of either the interrupted or continuous Lembert's suture. I have performed enterorraphy three times, though only once for gangrenous hernia, and in each case I have used Gély's suture, and I have found it on each occasion to have been perfectly efficient, but I should feel strongly tempted in my next case to employ Hagedorn's method, as being probably safer and possibly more rapidly applied.

However, we must bear in mind in discussing the question of the material to be used for suture and the method of applying it that these are matters of secondary importance. The point of importance above all others is not what one sews with, nor even how one sews, but the tissue into which we put our stitches. No sutures are good which are inserted in diseased parts. We should first make sure that we have removed all the suspicious portions of the intestine—better too much than too little. When this point is settled, next in importance is the care we exercise in putting in our sutures, whatever method we adopt. After these two, the question of the material we should employ sinks into significance, and it matters very little whether we use catgut or silk.

In conclusion, I think I am justified in summarising the points raised in this communication thus:

1st. The presence of ascitic fluid is not a common cause of strangulated hernia. It predisposes to umbilical hernia if the fluid is not too excessive; it tends to prevent strangulation at the inguinal or femoral rings.

2nd. Gangrenous hernia may be treated on one of two

[1] 'Brit. Med. Journ.,' April, 1892, p. 703.

[2] 'Medical Reprints,' Oct. 15, 1892, p. 133.

principles—either by resection of the affected portion and immediate suture, or the formation of an artificial anus.

3rd. The establishment of an artificial anus necessitates a secondary operation for its cure. This may be accomplished by secondary resection and suture, or by Dupuytren's method.

4th. It is illogical to compare the mortality following primary resection and suture with the mortality following secondary resection and suture, and thence to conclude that the establishment of artificial anus is preferable to primary resection and suture.

5th. To compare the death-rate of these two methods of treating gangrenous hernia, we should compare the death-rate following primary resection and suture with the death-rate following the formation of an artificial anus *plus* the death-rate of the secondary operation required for its cure.

6th. The mortality in cases of gangrenous hernia, treated by the formation of an artificial anus, is 80·7 per cent.

7th. The mortality of secondary resection and suture for the cure of artificial anus is 38 per cent.

8th. The mortality of treating an artificial anus by getting rid of the epéron (Dupuytren's method) is 7·3 per cent.

9th. The mortality which attends resection and immediate suture in gangrenous hernia is shown in the table of 222 published cases to be 47 per cent.

10th. The statistics show—(*a*) That intestinal resection and suture should be the operation of choice in gangrenous hernia; (*b*) that simple enterotomy, followed by the formation of artificial anus, should be reserved for absolutely special cases, and should be considered as an exceptional procedure.

11th. In excising a portion of intestine it is of the greatest importance to resect through healthy tissue.

Hence it is better to sacrifice too much than too little of the gut.

In the accompanying tables are recorded some details of 222 cases of gangrenous hernia treated by resection of the intestine and immediate suture; and I gladly take this opportunity of acknowledging with gratitude the assistance I have received in their compilation from Dr. T. E. Gordon of Dublin, and Mr. H. Stansfield Collier of St. Mary's Hospital. I am also indebted to the valuable paper by McCosh in the 'New York Medical Journal,' 1889, for the greater portion of the earlier cases.

Table of Two hundred and twenty-two Cases of Gangrenous Hernia treated by Resection and Immediate Suture of the Intestine.

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
1	Ramdohr, 1727	'Moebius disput. Anat.,' Gothingæ, 1751, vol. vi	Inguinal hernia	—	2 feet	Wedge removed	Invag. of upper end in lower; silk sutures	R.	—	—
2	De Vernale, 1728	'Observ. et remarq. de Chirurg. Pract.,' Mannheim, 1767	Do.	—	2½ inches	—	Invag. of upper in lower end; silk sutures	R.	—	—
3	Duverger, 1737	Louis, 'Mém. de l'Acad. roy. de Chir.,' 1757, p. 188	Do.	—	2 inches	—	Ends sewed with silk sutures over trachea of calf	R.	—	Trachea of calf passed per anum on 21st day.
4	Schmidt, Jos., 1759	'Dissertatio de Ileo,' Wien., 1759	—	—	6 inches	—	Silk sutures	R.	—	—
5	Nayler, 1794	Cooper, in 'Anat. and Treat. of Hernia'	Inguinal hernia	—	4 inches	Wedge removed	Silk sutures; mesen. held against abd. wound by sutures	R.	—	Fæcal fistula on 2nd day, which remained.
6	Cooper, A., 1806	Cooper on 'Hernia,' p. 147	Femoral hernia	—	2½ inches	—	—	D.	—	—
7	Do.	Loc. cit., p. 148	Do.	—	¾ inch	—	—	D.	Recovered from primary operation, but death in 2 months, due to closure of intestine	On 8th day fæcal fistula formed.
8	Diffenbach, 1836	'Wochensch. f. gesam. Heilkunde,' 1836, No. 26	Inguinal hernia	—	3 inches	Wedge removed	Lembert's suture (silk)	R.	—	Patient died in 4 weeks from intest. obstruct. well above sutures.
9	Von Langenbeck,	'Arch. kl. Chir.,' vol. xix, p. 410	Properitoneal	—	20 cm.	—	Invag. (Ramdohr), with separation of	D.	Delirium tremens and	Line of suture did not leak.

	1875		inguinal hernia				mesentery and Lembert's sutures of catgut		exhaustion	
10	Kuester, 1877	'Verh. der Gesellsch. f. Chir.,' 1879, p. 83	Femoral hernia	—	4 cm.	Wedge removed	Lembert's sutures, with catgut	D.	Septic peritonitis	Two sutures had given way, and leakage taken place.
11	Czerny, 1878	'Berl. klin. Wochensch.,' 1880, p. 638	Do.	F., 43	10 cm.	Do.	Two rows of silk sutures (24)	R.	—	—
12	Kuester, 1877	Report by Madelung in 'Arch. f. Chir.,' Bd. xxvii, p. 84	Ing. hernia. Before operation fæces had escaped in peritoneum	—	5 cm. by laparotomy	—	Lembert's suture (catgut)	D.	Septic peritonitis	8 hours after operation.
13	Kuester, 1878	Personal communication to Madelung	Inguinal hernia	—	5 cm.	—	Do.	D.	—	28 hours after operation.
14	Nicoladoni, 1879	'Wien. med. Blätter,' 1879, Nos. 6 and 7	Femoral hernia	—	—	—	Edges inverted, and silk sutures	R.	—	Fistula formed on 5th day, but afterwards closed.
15	Wahl, 1879	'St. Petersburgh med. Wochenschr.,' 1879	Pro-peritoneal inguinal hernia	—	15 cm.	Wedge removed	Lembert's suture, catgut (10)	D.	Peritonitis in 7 hours	Union of sutures good.
16	Woelfler	'Wien. med. Wochensch.,' 1881, p. 116	Femoral hernia	F., 65	15 cm.	—	25 silk sutures	D.	Septic peritonitis due to fæcal escape before operation	—
17	Hagedorn	'Verh. Gesellsch. f. Chir.,' 1880, p. 64	Do.	F., 68	16 cm.	Wedge removed	Lembert's suture of catgut	R.	—	On 5th day fæcal fistula formed.
18	Do.	Loc. cit., p. 65	Do.	F., 40	2 cm.	—	Do.	R.	—	On 6th day fæcal fistula.
19	Ludvik, 1880	'Wien. med. Presse,' 1880, p. 743	Do.	F., 60	10 × 12 cm. of ileum	Cut off close to attachment	Upper invag. in lower end; 20 catgut sutures	R.	—	Stool on 4th day.

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
20	Bardenheuer, 1880	'Die Drainirung der Peritonealhöhle,' 1881, p. 218	—	—	—	—	Two rows of sutures	R.	—	Fistula formed, which healed in 4 months.
21	Czerny	'Berl. kl. Wochensch.,' 1880, p. 45	Inguinal hernia	M., 49	6 cm.	Wedge removed	Czerny's	D. during operation	Collapse and impact of vomited matter in trachea	—
22	Heusner	'Dent. med. Wochenschr.,' 1880, p. 558	Femoral hernia	F. 73	10 cm.	Do.	Lembert's sutures, catgut (10)	D.	In 24 hours; septic peritonitis; leakage through line of suture	—
23	Gussenbauer, 1880	'Prager Wochenschr'	Inguinal hernia	—	18 cm.	Do.	Two rows suture, Lembert's (catgut)	R.	—	On 6th day fistula formed; healed on 14th day.
24	Julliard, 1881	'Rev. med. de la Suisse romande,' June 15, 1881	Femoral hernia	—	18 cm. by laparotomy	Do.	Jobert's method (catgut)	R.	—	—
25	Jaffé, 1881	'Samml. klin. Vorträge' (Hamburg), p. 1699	Do.	F., 52	12 cm.	Do.	Czerny-Lembert (catgut)	R.	—	Radical cure. Stool (spontaneous) on 4th day.
26	Taendler, 1881	Personal communication to Dr. Nebel. Rep. by Madelung	Do.	F, 52	11 cm.	—	Ten catgut sutures between serous and muscular coats	D.	In 62 hours after operation; septic peritonitis	Sutures loose; intest. gang. six inches above.
27	Billroth, 1881	Communicated by Woelfler. Rep. by Madelung, 'Arch. kl. Chir.,' Bd. xxvii, p. 277	Inguinal hernia	M., 60	5 cm.	—	Two rows silk; outer row in Lembert's method	R.	—	Fistula formed, which closed in 3 weeks.
28	Roggenbau, 1881	'Berl. kl. Wochensch,' 1881, No. 29	Do.	—	32 cm.	—	Czerny-Lembert (silk)	R.	—	Stool on 5th day.
29	Rydygier	'Berl. kl. Wochensch.,' 1881,	Femoral hernia	F., 58	54 cm.; project-	—	First row of contin. gut through	D.	Peritonitis in 20 hours;	—

		p. 593			ing ends of muc. mem. cut off		ser. and musc. coats; second row of Lembert's		line of suture closed	
30	Billroth (Woelfler)	'Verhand. d. Gesellsch. Chir.,' 8th Cong., p. 85	Do.	—	—	—	—	D.	—	—
31	Billroth	'Wien. med. Wochensch.,'1881, No.5 (Nittelshöfer)	Crural hernia	—	15 cm.	—	Two rows, 25 sutures	D.	Collapse	Fæces in perit. cav. probably escaped in pulling out intestine.
32	Billroth (Woelfler)	'Wien. med. Wochensch.,'1881, No. 5, (Nittelshöfer), p. 85	—	—	—	—	—	D.	—	—
33	Bryk	Schramm in 'Przeylad Lekarski,' 1881, No. 22. Mentioned by Rydygier	Femoral hernia	F., 36	12 cm.	—	Lembert's	D.	Collapse	In a few hours after operation.
34	Do.	Loc. cit.	Inguinal hernia	M., 29	12 cm.	—	Do.	D.	On 7th day from inflam. of lungs and carbolic acid poison	Patient vomited from time of operation until death.
35	Obalinski	Personal communication to Rydygier	No details	—	—	—	—	D.	—	—
36	Do.	Loc. cit.	Do.	—	—	—	—	D.	—	—
37	Do.	Loc. cit.	Do.	—	—	—	—	D.	—	—
38	Do.	Loc. cit.	Do.	—	—	—	—	D.	—	—
39	Korzeniowski	Loc. cit.	—	—	—	—	—	D.	—	—
40	Weiss	Communication to Michl. Mentioned by Rydygier	Inguinal hernia	F., 47	10 cm.	—	Ten Lembert and 12 superficial fine silk sutures and invagination	D.	Inanition due to fæcal fistula	Died at end of 4 weeks.
41	Nichaus, P.	'Correspondenzbl. Schweiz Aerzte,' 1886, p. 70	Femoral hernia	F., 58	—	—	—	D.	In 2½ days; due to inanition	—

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
42	Kofranyi	'Wien. med. Wochensch.,' 1885, p. 965	—	F., 50	—	—	Serous coat only united	R.	—	—
43	Rosenberger	'Munch. med. Wochensch.,' 1888, p. 447	Femoral hernia	F., 40	—	—	Serous coat only	R.	—	—
44	Jones, S.	'Lancet,' 1885, p. 616	—	F., 42	—	—	Czerny-Lembert	?	Patient died at end of 15 d.; supposed to be due to abortion; sept. peritonitis present	—
45	Viertel	'Deut. med. Wochensch.,' 1877, No. 10	Femoral hernia	F., 56	3 cm.	Wedge removed	Lembert, of silk (15)	R.	—	—
46	Fischer	Reported by Reichel, 'Deut. Zeitschr. Chir.,' vol. xix	Umbilical hernia	F., 55	5 cm.	—	Lembert's	D.	Septic peritonitis on 2nd day	—
47	Do.	Reichel, loc. cit.	Inguinal hernia	F., 47	7 cm.	—	Do.	R.	—	—
48	Molodenkow and Minim	'Centralbl. f. Chir.,' 1881, No. 46	Do.	M., 21	—	—	Lembert's catgut (25)	R.	—	Bowels moved 3rd day.
49	Roser	Loc. cit., No. 52	Femoral hernia	—	1½ cm.	—	Two rows; 8 in inner and 6 in outer; coats inverted	D.	In 42 hours from ileus. Valve-like swelling of mucous memb.	—
50	Aman	Schmidt's 'Jahrbuch,' 1882, p. 266	Do.	F., 63	—	Wedge removed	Czerny's	D.	In 17 hours, from peritonitis	Line of suture closed.
51	Bardeleben	'Deut. Gesellsch.	—	—	—	—	—	D.	—	No details.

		f. Chir.,' Berlin, 1883								
52	Do.	—	—	—	—	—	—	D.	—	Do.
53	Feld	'Arch. kl. Chir.,' Bd. xxx, p. 222	Inguinal hernia	M., 33	18 cm.	Wedge removed	Two rows of silk interr. sutures	R.	—	—
54	Hofmokl	'Wien. med. Presse,' 1888, pp. 357 and 394	Umbilical hernia	F., 48	22 cm.	—	Three rows	D.	Peritonitis at end of 24 hours	—
55	Reichel	'Deut. Zeitsch. f. Chir.,' Bd. xix, p. 230	Do.	F., 56	21 cm.	—	Lembert's	D.	Septic peritonitis of 2 days	Union imperfect; operation lasted 2½ hours.
56	Do.	Loc. cit.	Inguinal hernia	F., 55	5 cm.	—	Do.	D.	Septic peritonitis on 2nd day	Leakage at line of suture.
57	Do.	Do.	Do.	F., 47	7 cm.	—	Do.	R.	—	—
58	Do.	Do.	Do.	M., 35	37 cm.	—	Do.	R.	—	Fæcal fist., formed by punct. of intestine on account of obstruction.
59	Serres	'Rev. de Chir.,' 1887, p. 928	Do.	F., 69	8 cm.	—	—	R.	—	—
60	Prater	'Rev. de Chir.,' 1884, p. 859	Do.	F., 61	21 cm.	—	Two rows, 1 for muc. memb., and 1 for serous coats	D.	Shock at end of 24 hours	—
61	Ill, E. J.	'Med. Record,' Sept. 22nd, 1892	Femoral hernia	F., 24	—	—	Lembert's	R.	—	On 5th day fæcal fistula formed.
62 to 81	Mazzoni	'Ital. Cong. of Surg.,' 1886, reports 20 cases done in Italy up to 1886	19 cases of strangulated hernia	—	—	—	—	11 R.; 8 D.	—	—
81	Mazzuchelli, A.	'Gaz. Med. Ital.,' Lomb., 1884, p. 475	Inguinal hernia	M., 51	16 cm.	—	Czerny-Lembert	R.	—	Operation lasted 2 hours
82	Sobolewski	'Deut. med. Wochensch.,' 1887, p. 496	Femoral hernia	F., 26	15 cm.	Wedge removed	Two rows interrupted	R.	—	Operation lasted 1¼ hour

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
83	Nedopil	'Wien. med. Wochensch.,'1886, No. 51	Inguinal hernia	M., 52	8 cm.	Mesentery separated at attachment	Two rows; in part continuous, and in part interrupted	R.	—	—
84	Do.	Loc. cit.	Do.	F., 48	20 cm.	—	—	D.	From septic peritonitis on second day	Gangrene of intestine for 3 cm. on both sides of sutures.
85	Do.	Loc. cit.	Femoral hernia, which slipped into abdomen, and fæces escaped	F., 53	—	—	—	D.	In 6 hours from septic peritonitis and collapse	Laparotomy was performed; death not due to operation.
86	Do.	'Wien. med. Wochensch.,'1886, No. 51	—	F., 49	6 cm.	—	Two rows of interrupted sutures	R.	—	—
87	Burckhardt	'Correspondenzbl. f. Schweiz. Aerzte,' 1887	Inguinal hernia	F., 21	—	—	Inner row continuous; outer row interrupted	R.	—	—
88	M. Banks	'Lancet,' 1885, p. 750	Do.	M., 25	—	Wedge removed	Czerny-Lembert; catgut	R.	—	Operation lasted 2 hours
89	Parker	'Brit. Med. Journ.,' Jan. 22nd, 1887	Do.	M., 50	12 inches	Do.	Two rows of inter. catgut; mucous membrane not included	R.	—	—
90	Carson, N. B.	'Journ. Amer. Med. Assoc.,' May 7th, 1887	Inguinal hernia; laparotomy	M., 12	2½ inches	Do.	—	R.	—	—
91	Stewart, J. E.	'Amer. Journ. of Med. Sci.,' 1886, p. 152	Femoral hernia	M., 68	4 inches	Removed at point of attachment	Lembert's, of catgut	R.	—	—

92	Crespi	'Bollett. Cliniche,' Milano, Sept., 1887	Inguinal hernia	F., 53	—	—	Lembert's	R.	—	—
93	Postempski	'Bollett. dell' Acad. Med. di Roma,' Dec., 1887	Inguinal hernia, right	M., 20	—	—	—	R.	—	—
94	Prati	'Centralbl. f. Chir.,' 1883, p. 45	—	—	—	—	—	D.	Shock a few hours after operation	—
95	Marcy	Rep. by Ill; loc. cit.	—	—	—	—	—	D.	—	—
96	Rosenberger	'Magdeburg Naturversamm.'	Inguinal hernia	—	—	—	—	D.	—	—
97	Do.	Loc. cit.; rep. by Hænel, 'Arch. kl. Chir.,' Bd. xxx, p. 395	Femoral hernia	—	—	—	—	D.	—	—
98	Kosinski	'Centralbl. f. Chir.,' 1886, p. 173	—	—	—	—	—	D.	—	—
99	Do.	Loc. cit.; rep. by Hænel	—	—	—	—	—	D.	—	—
100	Riedel	'Deut. med. Wochensch.,' 1883, p. 656	Inguinal hernia	—	30 cm.	—	—	D.	In 4 days; septic peritonitis	Intest. gang. above and below sutures
101	Wettergren	'Hygeia,' 1883; 'Centralb. f. Chir.,' 1883, p. 784	Femoral hernia	F., 60	10 cm.	—	Catgut	D.	Septic peritonitis in 2 hours	In replacing gut fæces escaped in abdomen.
102	Do.	Loc. cit.	Do.	F., 65	10 cm.	Wedge removed	—	R.	—	Death in 25 days from cancer.
103	Walter, K. A.	'Hygeia,' 1886; 'Centralb. Chir.,' 1886, p. 692	Inguinal hernia	M., 52	28 inches	Separated at attachment	Two to three rows of Lembert's suture	R.	—	—
104	Agnew, D. H.	'Med. and Surg. Reporter,' 1888, p. 321	Umbilical hernia	F.	14 inches	Wedge removed	Lembert's silk	D.	In 24 hours from hæmorrhage	1 suture torn out, but no leakage.
105	Fuller	'Med. Rec.,' vol. xxii, p. 430	Femoral hernia	F., 50	5½ inches	—	Continuous linen	R.	—	—

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
106	Richardson, M. H.	'Trans. Amer. Surg. Assoc.,' May, 1889	Umbilical hernia	F., 42	8 inches	Ligated with silk	Single row—Lembert	R.	—	—
107	Do.	Loc. cit.	Inguinal	F., 65	—	Continuous silk suture	Interrupted Lembert silk	D.	Exhaustion or collapse in 12 hours	—
108	Rosenberger, J. A.	'Münch. med. Wochensch.,' 1888, p. 447	Femoral hernia	F., 40	—	—	Serous coat only sutured	R.	—	—
109	Burtscher	'Correspondenzbl. Schweiz. Aertze,' 1881	Do.	F., 40	—	—	—	D.	On 5th day	—
110	Lange, F.	Personal communication to McCosh	Inguinal hernia	M., 45	5 inches	—	Czerny-Lembert	D.	Collapse in few hours after operation	—
111	Briddon, C. K.	Do.	Femoral hernia	F., 40	4 inches	—	Lembert's	D.	Collapse within 24 hours after operation	—
112	McCosh, A. J.	'New York Med. Journ.,' vol. xlix, 1889, p. 281	Inguinal hernia	F., 66	4 inches	Wedge removed	Czerny-Lembert; inner of silk, outer of catgut	R.	—	—
113	Feld	Klaussner's paper in 'Münch. med. Woch.,' 1889	Left inguinal	M., 33	18 cm.	Small V-piece removed, and sewn with 4 "button" sutures	28 Lembert's sutures	R.	—	Had an attack of pneumonia shortly afterwards, but discharged cured in a month.
114	Hagemann	'Deutsche med. Woch.,' No. 31, 1889, p. 629	Right femoral	F., 46	14 cm.	V-piece of mesentery removed	Two rows of sutures; (1) of mucosa, (2) of musculat and serosa	R.	—	—

115	Postemski	'Bull. r. d. Med. di Roma,' 1886, vol. xii	Inguinal (? side)	F., 54	17 cm.	Divided close to gut, and its edge sutured	Prolapsed mucosa cut away. Catgut "mantuamaker's" hem of all the coats, and a Lembert suture of a second row. Lot. Hyd. Perchl., 1 in 1000	R.	—	This is the 5th case reported at Rome, all successful.
116	Duranti	Loc. cit.	Right inguinal	M., 18	35 cm.	V-piece exsected	Two rows of sutures. "Ippolito Palesciano mode," through muscle and serous layer	R.	—	One of the 5 cases mentioned above.
117	Gay	'Boston Med. and Surg. Journ.,' 1892, p. 207	Umbilical	F., 60	13 cm.	?	Two rows of silk continuous sutures. (1) Through muscle and serous. (2) Through serous alone	D. on 5th day	Fæcal extravasation at junction of gut with mesentery	Gangrenous omentum removed.
118	Do.	Loc. cit.	Do.	F., 55	More than 15 cm.	? Nearly certainly V-piece removed	Do.	R.	—	The case was complicated by large fibroid and ascites. The mesentery opposite the gangrenous gut was also gangrenous.
119	Bozella	'Gazetta med. Lombard.,' 1891 p. 514	Right femoral (Richter's partial)	F., 54	6 cm.	V-piece removed	No. 1 Lister's catgut "Rem" of mucosa alone, and a second suture, also continuous, of serosa and muscle	R.	—	Patient, had to complicate diagnosis, a cyst in the right labium majus. No complications.

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
120	Jaboulay	'Lyon Medical,' 1891, p. 436	Right femoral	M., 60	10 cm.	?	Gussenbauer's suture of silk. Lower opening of intestine smaller than upper, and therefore a small longitudinal slit was made to render the former equal to the latter	R.	—	—
121	Ransohoff	'Journ. Amer. Med. Assoc.,' Chicago, 1892, p. 198	Right inguinal	F., 56	35 cm.	Divided a short distance from gut, and united with a continuous suture	Fine silk "continuous Lembert," with additional points where they appeared to be wanted	R.	—	—
122	Klaussner	'Munchen. med. Woch.,' 1889	Umbilical	F., 34	5 cm.	Probably Kocher's method	Fine silk, Lembert's suture	D. From shock next day	—	Patient was in 7th month of pregnancy, and was in bad state at time of operation. Gangrenous omentum removed.
123	Do.	Loc. cit.	Do.	F., 61	40 cm.	Wedge removed	Two rows of fine silk—(1) of mucosa, (2) of serosa	R.	—	—
124	Kohler	Loc. cit.	Right inguinal	M., adult	"Large loop"	Probably V removed	Two rows of continuous suture—(1) of mucosa, (2) of serosa	R.	—	—

125	Anderegg	Loc. cit.	—	F., 43	12 cm.	Kocher's method	Eighteen points of catgut by Lembert's method	D. From collapse 8 hours after operation	General peritonitis	Perforation had occurred, producing intraperitoneal extravasation before operation.
126	Do.	Loc. cit.	—	F., 42	12 cm.	—	Immediate suture, mode not stated	D. From collapse 36 hrs. after operation	No P.M. allowed	The sutured intestine was fixed in wound.
127	Jaboulay	'Lyon Medical,' 1891, p. 436	Right femoral	M., 40	12 cm.	?	Gussenbauer's suture, as in Case 120	Local recovery. Died from gangrenous pneumonia 2 months later	Perfect union. Seat adherent to parietes and to another loop of gut. Silk still intact	"Jaboulay prefers Gussenbauer's suture as being more rapidly accomplished than Czerny-Lembert's."
128	Dawborn	'New York Med. Record,' 1889, vol. xxxv, p. 428	Left femoral	F., 55 "(at least)"	31 cm.	Catgut "cobbler's" suture at ⅜ inch from gut and mesentery cut close to this line	Two rows of sutures, a Czerny and a Lembert catgut. The row of Lembert's sutures "not drawn too tightly," and the sutures of first row alternating with those of second row. Irrigation with warm salt solution	R. (But case reported between 2 and 3 weeks after operation)	—	"This method of dealing with mesentery puckers it so that it is greatly shortened."

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
129	Park	'New York Med. Journ.,' 1889, vol. 50, p. 603	Umbilical	F., 39	Not whole circumference	Not touched	No. 1 catgut; three rows of continuous suture—(1) through mucosa; (2) through muscle; (3) through serosa	R. Well 6 mos. after	—	An ellipse to include two gangrenous spots ¼ inch apart, close to mesentery. 3 months after operation symptoms lasting 12 hours and suggesting intestinal obstruction, but passing off and not recurring.
130	Hagemann	'Deutsche med. Woch.,' 1889, No. 31, p. 627	Left inguinal; congenital	M., 12	31 cm.	Wedge-shaped piece exsected	(1) Suture of mucosa; (2) a row of Lembert's	R. Well a year after	—	—
131	L. Frey	'Wien. med. Presse,' 1888, vol. xxix, p. 1484	Right inguinal	M., 28	33 cm.	—	Two rows—(1) continuous suture "after Rydygier;" (2) interrupted suture	R.	—	Gangrenous omentum removed.
132	Cheever	'Boston Med. and Surg. Journ.,' vol. cxvii, 1889, p. 8	Right congenital	M., 20	9 cm.	"Mesentery secured by a running suture"	A dozen Lembert's stitches	D. From unrelieved obstruction 84 hours later	A volvulus undiscovered at operation below hernia, and a foot above cæcal valve. Union good except at one point, where edge appeared to have sloughed	The volvulus was bowel sharply turned on itself, thrust like a finger into right side of pelvis, and fixed by adhesions.
133	H. A.	'Philadelphia	Femoral	F.,	5 cm.	Not	Divided ends	D.	No autopsy	Walker says:—"I

	Walker	Med. and Surg. Reporter,' 1887, vol. lvii, p. 853		32		given	sutured with continuous catgut suture	From peritonitis 4th day		feel confident that had my antiseptic precautions been perfect in this case the results would have been different."
134	Croskey	'Philadelphia Times and Register,' 1889, p. 223	Right inguinal	M., 29	13 cm.	"Mesentery united by catgut"	Black silk Czerny-Lembert, with additional row of Lembert sutures	R.	—	Gangrenous omentum removed.
135	Moreau	'Bull. d'Ac. Med. de Belge,' 1891, p. 674	Do.	M., 33	24 cm.	—	Carbolised silk, No. 1; 20 Lembert's stitches	R.	—	—
136	Clarke Stewart	'Internat. Journ. Med. Sci.,' vol. xci, 1886, p. 152	Right femoral	M., 68	4 inches	?	Lembert's	R.	—	Large soft stool on 5th day.
137	Arbuthnot Lane	'Trans. Clin. Soc. London,' vol. xxiv, 1891, p. 182	Do.	F., 53	3 inches	Ligatured	Senn's plates and Lembert's suture	R.	—	Left hospital in 3 weeks and 4 days.
138	Do.	Loc. cit.	Do.	F., 55	—	?	Senn's plates and omental graft	D.	Gangrene of proximal end of gut	—
139	Martinet	'Rev. de Chirurgie,' vol. ix, 1889, p. 335	Femoral	F., 58	10–12 cm.	?	Gély's suture	R.	—	On 22nd day abscesses formed, but cure complete after several months.
140	Gerster, A. G. (operation, 1881)	'Aseptic and Antiseptic Surgery,' 1888	Umbilical transverse colon	F., 61	6 inches	Wedge of meso-colon	Fine catgut	D.	Peritonitis	—
141	Surgeon-Major W. R. Browne	'Indian Medical Gazette,' March, 1890, p. 89	Right inguinal	?	?	Wedge removed	Lembert; fine catgut	R.	—	Radical cure. Bowels moved 48 hours after operation.
142	T. N. Fitzgerald (operation, 1879)	'Australian Med. Journal,' 1883, N. S., vol. v, p. 31	Do.	M., 55	?	Do.	Catgut	D.	Leakage at mesenteric border. Peritonitis	Death in 67 hours.

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
143	Do. (operation, 1882)	Loc. cit.	Inguinal	M., 56	?	Do.	Continuous catgut, "herring-bone"	D.	Collapse	Died 5 hours after operation.
144	J. A. Wyeth	'New York Med. Journ.,' March 19, 1887	Femoral	F., 56	2½ inches	Do.	Czerny's suture through mucous memb.; Lembert's, three rows	R.	—	Radical cure.
145	Luzenberg, C. A.	'Gross. System of Surgery,' 1882, vol. ii, p. 533	?	?	6 inches	?	?	R.	—	—
146	Rushton Parker	'Med. Times and Gaz.,' vol. i, 1882, p. 581	Right inguinal	M., 60	12 inches	Gangrenous portion excised	Catgut; interrupted and continuous	D.	Septicæmia	Death in 36 hours. Union found perfect.
147	Do.	'Liverpool Med.-Chir. Journ.,' Jan., 1886, p. 234	Umbilical	F., 52	6 inches	?	Catgut; Glover's stitch	D.	Collapse	Death in 12 hours.
148	Lücke	'Sonnenberg Deutsche Zeitsch. für Chirurgie,' 1873, vol. xii, p. 311	Femoral	F., 54	6 cm.	—	Ramdohr's invagination; double row sutures	D.	Peritonitis	Tendency to stricture at seat of invagination. Death same day.
149	G. Bouilly	'Revue de Chirurgie,' vol. iii, 1887, p. 362	Right inguinal	M., 47	50 cm.	Gangrenous portion excised	Lembert; fine catgut	D.	Acute peritonitis	Spreading gangrene. Secondary operation.
150	R. Gattai	'Lo Sperimentale,' Jan., 1882, p. 54	Right femoral	F., 67	1½ cm.	—	—	R.	—	—
151	Rochelt	'Wien. med. Presse,' No. 36, 1882	Right inguinal	M., 54	12 cm.	Gangrenous portion excised	Czerny-Lembert	D.	Collapse in 8 hours	—
152	G. Bouilly	'Revue de Chirurgie,' vol. iii, 1883, p. 362	Left femoral	F., 50	21 cm.	Corresponding piece excised	Lembert, silk	D.	Kinking of intestine above line of suture	27 hours after operation. Line of union found perfect.

153	Von Lukowicz	'Archiv. für klin. Chir.,' Band 42, 1891, p. 491	Right inguinal	F., 54	5½ inches, duodenum	Corresponding piece of mesent. excised	Lembert, two rows silk	R.	—	On 4th day spontaneous stool.
154	Von Baracz (operation Aug., 1886)	Loc. cit., pp. 493—522	Do.	M., 41	36 cm.	Resected crossways	First row, Czerny of mucosa; second row, Lembert	R.	—	Fæcal fistula persisted. 6 months later secondary operation, with excision of 6 cm. more. Recovery. Digital dilation of efferent end of gut.
155	Kocher (operation Feb. 18, 1879)	'Deutsche Zeitschrift für Chirurgie,' Band 32, 1891, p. 101	Right femoral	F., 49	8 cm.	?	Twenty-five catgut	D.	Perforation of intestine below seat of suture	—
156	Do. (op. March 5, 1880)	Loc. cit., p. 103	Left inguinal	F., 49	42 cm.	—	Silk, Lembert	R.	—	—
157	Do. (May 19, 1880)	Loc. cit., p. 106	Right femoral	F., 61	11 cm.	—	Eight Lembert's silk; second row	D.	Peritonitis; pulmonary embolism	Death in 8 days. Littré's hernia.
158	Do. (July 6, 1880)	Loc. cit., p. 108	Right femoral	F., 31	21 cm.	—	Eight catgut Lembert, and a second row	D.	—	Gangrene after *reposition en bloc.*
159	Do. (Nov. 26, 1880)	Loc. cit., p. 110	Right inguinal	M., 34	12 cm.	—	Fifteen catgut	D.	Peritonitis; broncho-pneumonia	—
160	Do. (Feb. 10, 1881)	Loc. cit., p. 113	Do.	M., 27	25 cm.	Gangrenous portion excised	Twenty Lembert's	D.	Previous peritonitis	—
161	Do. (July 21, 1881)	Loc. cit., p. 115	Right femoral	M., 49	8 cm.	Involved portion excised	Twenty-five Lembert's	R.	—	Radical cure.
162	Do. (Dec. 15, 1881)	Loc. cit., p. 117	Do.	M., 42	7 cm.	Necrotic portion excised	Two rows of suture	R.	—	Littré's hernia.

No	Operator.	Where reported.	Disease.	Age and sex	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
163	Kocher (Jan. 26, 1882)	'Deutsche Zeitschrift für Chirurgie,' Band 32, 1891, p. 120	Do.	F., 60	25 cm.	—	—	D.	Peritonitis	Frequent and prolonged attempts at taxis before operation.
164	Do. (Feb. 10, 1885)	Loc. cit., p. 122	Do.	M., 18	—	—	Two rows of suture	D.	Purulent peritonitis	No leakage.
165	Do.	'Correspondenzbl. für Schweiz-Aertze,' 1886	Inguinal	M., 57	1 m., 60 cm.	Separated close to attachment to gut	Inner interrupted, outer continuous. Both silk	R.	—	Out of bed on 14th day; discharged on 18th day. No bad effects.
166	Do. (Apr. 22, 1886)	'Deutsche Zeitschrift für Chirurgie,' Band xxxii, 1891, p. 131	Right femoral	M., 65	—	Divided parallel to intestine	First row silk through whole thickness of intestine; second row serous and continuous	D.	Partial gangrene of intestine; acute sepsis	Death 15 days after operation.
167	Do. (June 9, 1886)	Loc. cit., p. 138	Do.	M., 39	8 cm.	Do.	Do.	R.	—	Hernia returned in 1889. Littré's.
168	Do. (Oct. 22, 1887)	Loc. cit., p. 139	Left femoral	F., 46	21 cm.	Portion excised	Two rows continuous	R.	—	Radical cure.
169	Dr. Lardy, First Assist. (May 26, 1888)	Loc. cit., p. 144	Right femoral	M., 50	10 cm.	—	First row interrupted; second row continuous	R.	—	Well in 16 days. Littré's.
170	Kocher (June 17th, 1888)	Loc cit., p. 145	Do.	F., 34	4½ cm.	Divided parallel to intestine	Lembert's continuous	R.	—	Littré's.

171	Do. (July 10, 1888)	Loc. cit., p. 146	Right inguino-properitoneal	M., 19½	3 cm.	—	First row through whole thickness of intestinal wall; second row continuous, Lembert	R.	—	Littré. No return of hernia in 12 months.
172	Do. (Aug. 11, 1888)	Loc. cit., p. 153	Left femoral	F., 77	20 cm.	—	First row interrupted; second row continuous	D.	—	Some leakage; 15 cm. of intestine in a semi-gangrenous condition.
173	Do. (Sept. 19, 1888)	Loc. cit., p. 156	Left inguinal	F., 62	28 cm.	—	Do.	D.	—	—
174	Hagedorn (Jan., 1886)	'Deutsche Zeitschrift für Chirurgie,' Band xxxii, pp. 323—376	Right inguinal	M., 48	12 cm.	In all Hagedorn's cases, he ligatures the mesentery parallel to the intestine, about 2 cm. from the gut, with silk or elastic ligatures. He begins to ligate at a point in mesentery corresponding to middle of intestinal loop to be excised, and ends at a point 1 cm. before reaching the place where the intestine is to be divided. Mesentery is then divided along whole length of its attachment to the loop to be excised	Catgut—first row continuous through mucosa and muscularis; second row continuous, Lembert	R.	—	Small fæcal fistula on 8th day, which soon closed. Firmly healed on 48th day.
175	Do. (May, 1890)	Loc. cit.	Right inguinal	M., 61	38 cm.	Do.	Do.	R.	—	No radical cure.
176	Do. (March, 1885)	Loc. cit.	Do.	M., 68	10 cm.	Do.	Do.	D.	Purulent peritonitis existing before operation	No leakage. Place of suture withstood test by water.

No	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery	Suture material.	Result.	Cause of death.	Remarks.
177	Hagedorn (Dec., 1885)	Loc. cit.	Do.	M., 73	24 cm.	Do.	Do.	D. in 20 hours	Collapse	Extensive carcinoma, cardiæ ventriculi, and of the peritoneum found. Serosa found glued firmly; withstood water test.
178	Do. (June, 1888)	Loc. cit.	Right femoral	M., 88	4 cm.	Do.	Do.	R.	—	—
179	Do. (Nov., 1884)	Loc. cit.	Do.	F., 84	9 cm.	Do.	Do.	R.	—	Radical cure.
180	Do. (Feb., 1886)	Loc. cit.	Do.	F., 53	20 cm.	Do.	Do.	R.	—	Do.
181	Do. (Dec., 1889)	Loc. cit.	Do.	F., 54	10 cm.	Do.	Do.	D. in 48 hours	—	Union firm. Withstood the water test.
182	Do. (Nov., 1889)	Loc. cit.	Do.	F., 55	11 cm.	Do.	Do.	D. in 36 hours	Fatty degeneration of heart. An inebriate	Do.
183	Do. (Oct., 1889)	Loc. cit.	Do.	F., 59	12 cm.	Do.	Do.	D. in 22 hours	—	Do.
184	Do. (Dec., 1885)	Loc. cit.	Do.	F., 66	17 cm.	Do.	Do.	D. in 12 hours	Pre-existing anasarca	Closure of wound in intestine was perfect, and withstood water test after 12 hours
185	Do. (Feb., 1889)	Loc. cit.	Left femoral	F., 48	7 cm.	Do.	Do.	R.	—	In 19 days.

186	Do. (Dec., 1884)	Loc. cit.	Do.	F., 67	13 cm.	Do.	Do.	R.	—	Patient had perfectly recovered, when she died of heart disease and pneumonia on 27th day.
187	Do. (April, 1888)	Loc. cit.	Do.	F., 38	12 cm.	Do.	Do.	D. in 9½ hours	Hæmorrhage from a vessel in mesentery	—
188	Do. (July, 1888)	Loc. cit.	Umbilical	M., 88	8 cm.	Do.	Do.	D. in 10 mins.	Peritonitis and septicæmia	Patient moribund at time of operation. Hernia 14 days strangulated.
189	Do. (May, 1890)	Loc. cit.	Do.	F., 64	43 cm.	Do.	Do.	D. in 38 days	Acute purulent peritonitis, due to elastic ligature	Patient left perfectly well on 27th day. Peritonitis subsequently developed.
190	Cohn	'Berlin. klin. Wochensch.,' 1889, Nos. 20 and 21	Right crural, partial (Richter?)	F., 24	6 cm.	—	A row of sutures of mucosa, and a row of Lembert's sutures	D. in 18 hours	Septic peritonitis	Bowel was torn, and fæces escaped into peritoneal cavity. Incision was prolonged up to the umbilicus, and abdomen washed out with thymol solution.
191	Do.	Loc. cit.	Right crural	F., 31	34 cm.	—	A row of sutures of mucosa, and a row of Lembert's. Bowel left in the sac	D. on 2nd day	Suppurative peritonitis. A leak at one spot	Fæces came out between the sutures of gut on the first day.
192	Do.	Loc. cit.	Left inguinal	M., 57	6 cm.	—	A row of mucosa stitches, and a row of Lembert's	D. on 3rd day	Broncho-pneumonia	Sutures holding well; no peritonitis. Patient was a weakly man, suffering from chronic bronchitis.

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
193	Cohn	Loc. cit.	Left femoral	F., 59	20 cm.	—	Probably as in last case	R. after 5 weeks	—	An incision in linea alba was made for the suturing. Fæcal fistula, which closed after a time spontaneously.
194	A. Casini	'Giorn. Internaz. della Scienza med.,' Napoli, 1889, p. 355	Left inguinal	F., 40	18 cm.	V-piece excised	Continuous fine-silk suture of all the tunics for three quarters of the circumference, and the circle at the mesenteric part completed by Apolito's suture. (2) A second row of Lembert's stitches	R.	—	Patient was the subject of bronchial catarrh, and had an acute attack soon after operation.
195	Dietrichsen	'Norsk. Mag. f. Lagevidensk.,' 1888, p. 487	Direct inguinal	M., 76	14 cm.	V-piece of mesentery removed	Wöeffler's method. Carbolised sutures. A row of Lembert's stitches	R.	—	This operation was done under cocain (6 per cent.). Gangrenous omentum was removed. Sac was gangrenous and gut ruptured. Astercoral fistula formed six days later, but it subsequently closed, and patient was well 8 months later.
196	Ström	Loc. cit., 1886, p. 573	Crural	F., 40	12 cm.	V-piece excised, and edges stitched with catgut continuous suture	"Czerny Lembert's double circle of iodoform silk stitches," but continuous, with here and there a knot	R.	—	There was a second constriction of gut, with doubtful vitality, and so when the sutured part was returned, it was decided to place "an iodoform silk sling round this doubtful part, through its mesentery, to fix it near the ring." This was removed at first dressing. No complications after operation.
197	Ferrajoli	'Giorn. Internaz. delle Sc. Med.,' Napoli, 1889,	Right crural	F., 40	15 cm.	V-piece excised	"Apolito's method." Continuous sutures,	D. on 14th	Bronchitis and diarrhœa. A very small	Gangrenous omentum excised. Sac was sloughy at

		p. 169					two, each taking half a circumference, and ending at mesenteric junction. Reinforced where necessary by fine catgut	day	localised peritoneal abscess surrounded by adhesions near place of suture. A second larger abscess the size of a small apple, between layers of mesentery near suture line. Perfect union of sutures. Chronic intestinal nephritis. Bronchitis and lung collapse	time of operation. Patient had long suffered from bronchitis.
198	Mickulicz	'Berlin. klin. Wochenschrift,' 1892, Nos. 10—13	Right femoral	F., 36	32 cm.	Kocher's	Two rows silk Czerny-Lembert. Hagedorn's needle-holder. Gussenbauer's clamp	R.	—	Tamponade of femoral canal with iodoform gauze.
199	Do.	Loc. cit.	Do.	M., 51	90 cm.	Do.	Do.	D. in 30 hours	Acute peritonitis. Edges bluish-black for 5 cm. from suture	Gut had perforated before operation.
200	Do.	Loc. cit.	Right inguinal (Richter)	M., 46	3 cm.	Do.	Do.	R.	—	Tamponade with iodoform gauze.
201	Do.	Loc. cit.	Right femoral	F., 45	15 cm.	Do.	Do.	R.	—	The sutured bowel, before being dropped back, was surrounded with iodoform gauze.
202	Do.	Loc. cit.	Do.	F., 56	25 cm.	Do.	Do.	R.	—	Patient had serous diarrhœa, after a right-sided pleurisy.
203	Do.	Loc. cit.	Left inguinal	M., 39	—	Do.	Do.	D. in 10 hours	Peritonitis	Gangrenous part had ruptured, and gone back into abdomen. Incision was prolonged to umbilicus.

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
204	Mickulicz	Loc. cit.	Right femoral	M., 58	20 cm. (nearly)	Do.	Do.	D. from shock 5½ hours later	No peritonitis. Atheroma of aorta. Old pleurisy. Lungs congested	Omentum resected. Pulse was good, and no shock of importance when patient was put back to bed. End was unexpected.
205	Do.	Loc. cit.	Left femoral	F., 15	11 cm.	Do.	Czerny-Lembert, silk	R.	—	There was perforation before operation. Patient had left-sided pleurisy a few days after operation.
206	Do.	Loc. cit.	Right femoral (Richter)	F., 26	7 cm.	Do.	Do.	R.	—	Puncture through abdominal wall relieved distension by gas of intestine above lesion after operation.
207	Do.	Loc. cit.	Right femoral	F., 50	83 cm.	Do.	Do.	D. in 24 hours	Diffuse peritonitis	Pre-existing perforation.
208	Do.	Loc. cit.	Left femoral	M., 37	33 cm.	Kocher's	Do.	D. on 4th d., with reappearance of obstruction signs	"Fibrinous" slight (local?) peritonitis; well marked meteorism. Sutures holding	Do.
209	Do.	Loc. cit.	Do. ("Richter")	F., 57	10—12 cm.	Do.	Do.	R.	—	Slight symptoms of iodoform poisoning.

210	Do.	Loc. cit.	Right femoral	F., 35	25 cm.	Do.	Do.	D. on 7th day, with signs of peritonitis	Two small perforations 2½ cm. above place of suture. Suppurative peritonitis. Sutures held	Patient apparently doing well for first 24 hours.
211	Do.	Loc. cit.	Do.	F., 61	30 cm.	Do.	Do.	R.	—	—
212	Do.	Loc. cit.	Do.	F., 48	12—15 cm.	Do.	Do.	D. 14 days after	Hæmoptysis Adhesions round healthy suture line. Phthisis of both lungs	Had had phthisis for a year.
213	Do.	Loc. cit.	Do.	M., 50	33 cm.	Do.	Do.	D. in 24 hours	Peritonitis	Pre-existing perforation.
214	Do. (?)	Loc. cit.	Right inguinal	F., 45	16 cm.	Kocher's	Czerny-Lembert, silk	R.	—	At first 10 cm. were removed, and suture done, but seeing asphyxial state of suture line, 6 cm. were further removed.
215	Do. (?)	Loc. cit.	Right femoral	F., 37	30 cm.	Do.	Do.	D. in 24 hours	Purulent peritonitis. Sutures had given way in one place	Pre-existing perforation. An opening was made for washing out purulent contents of abdomen at time of operation.
216	Do. (?)	Loc. cit.	Left femoral	M., 38	30 cm.	Do.	Do.	R.	—	On the 9th day a small fæcal fistula was discovered, which remained open for 7 days, and healed, having discharged a silk suture.

No.	Operator.	Where reported.	Disease.	Age and sex.	Length removed.	Mesentery.	Suture material.	Result.	Cause of death.	Remarks.
217	Mickulicz (?)	Loc. cit.	Left inguinal	M., 23	40 cm.	Do.	Do.	R.	—	Pre-existing perforation.
218	Do. (?)	Loc. cit.	Left femoral	F., 44	90 cm.	Do.	Do.	R.	—	—
219	Potozki	'Proceedings Russ. Soc. of Phys.,' 1889, p. 196	? Inguinal	M., 30	32 cm.	—	Single row of Lembert's suture of fine silk	R.	—	—
220	Carl Beck	'New Yorker med. Monatsch.,' 1892, p. 249	Right inguinal	M., 45	12·5 cm.	Not sutured, but only folded	Double row of Czerny's suture (iodoform silk)	R.	—	—
221	Do.	Loc. cit.	Umbilical	F., 57	? (But quite a small piece)	Do.	Double row of Czerny's iodoform silk, and fixing to neck of sac by two sutures	R.	—	Fæcal fistula on 4th day. Secondary resection done 14 weeks later, and 9 cm. of gut removed; the same stitching done, and the same fixing at ring of sac. Discharge cured three weeks later.
222	Kendal Franks	Present communication	Do.	F., 30	23½ cm.	Wedge removed. Sutured with fine catgut, continuous	Gély's, modified. Fine silk	R.	—	Ovarian tumour and ascites present; tumour removed 5 weeks later. Recovery.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 82.)

ACUTE RENAL DISLOCATION.

BY

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Now that movable kidneys are so often submitted to surgical interference, our knowledge of the pathological significance of certain clinical conditions which are observed in connection with them has become much extended, and enables us to differentiate the acute from the more chronic forms of the affection. It is to the former of these two conditions that I have ventured to give the above-named title; for though it has been referred to by some writers during the past few years, both in Germany and France, it has not received that general recognition in this country which it deserves.

Briefly stated, this set of symptoms may be said to consist of a series of acute attacks of pain occurring at longer or shorter intervals in an individual, one or both of whose kidneys are abnormally movable. These attacks usually supervene upon some violent exertion, and each attack seems to render the individual more prone to a repetition of these paroxysms of pain; less and less provocation as time goes on being required to produce them, until eventually life is only tolerable in a recumbent position. Sometimes a different course of events occurs, and the chronic movable kidney, with its dull, aching

pains, supervenes upon the acuter variety, and takes its place.

One of these attacks usually begins without any warning. The patient is seized with an acute pain in one or other of his kidneys. Like most pain of renal origin, it is very liable to radiate down the thigh and into the groin, or may be referred to some portion of the lower part of the abdomen. If the attack is a violent one the patient is at once incapacitated, and occasionally vomits almost instantaneously. In an hour or two the region of the affected kidney both in front and behind becomes acutely tender, and a local distension of the intestine often ensues. More rarely this distension spreads to the whole abdomen, often giving rise to a suspicion of peritonitis, and seeming to point for a while possibly to a sudden perforation of the intestine. But the difficulty of diagnosis, if it exists at all, soon clears up. If an examination of the abdomen can be obtained within an hour or an hour and a half after the onset of the symptoms, and before the distension of the intestine has taken place, much difficulty is not usually experienced in arriving at a correct interpretation of the symptoms. Some enlargement of the kidney can generally be detected, and pressure of the tumour gives rise to a peculiar sensation of nausea, occasionally passing on to actual vomiting, and often accompanied by faintness. But before long a period of obscurity supervenes; the abdomen, which was lax and painless, grows tumid, flatulent, and agonising when handled, and at times a cold sweat stands on the brow of the sufferer. This condition, in which pain is the prominent symptom, may remain almost unchanged for several days, but it usually begins to subside after some hours, and after a few days the kidney regains its abnormal mobility, of which perhaps the attack in question has afforded the first indication either to the patient or his medical attendant. The condition of the urine during these attacks is subject to considerable variation, dependent probably, as we shall see later on, upon the extent to which the blood-

supply is interfered with. It may be scanty and blood-stained or almost porter-coloured, and smoky, and bearing a marked resemblance to the urine of acute Bright's disease. When it occurs, one of the first and most reliable symptoms of the abatement of the attack is the passage of a considerable amount of clear, pale urine of a very low specific gravity (so-called hysterical urine). Occasionally these attacks are accompanied by marked pyrexia and general constitutional disturbance, and when this is the case there is some difficulty in distinguishing them from more grave and serious affections.

I have ventured to give to this set of symptoms the name "acute renal dislocation" in preference to "renal incarceration," because it has seemed to me to express concisely the actual course of events by which this condition is produced. It is a condition, I believe, which is more closely allied to that which occurs when the head of a bone is displaced from its position than to those conditions which lead up to and follow upon strangulated hernia. The real difference probably between the ordinary movable kidney and the dislocated organ which gives rise to these acute attacks depends on the fact that the latter becomes at once fixed in its newly acquired position, which it only assumes in consequence of some violent strain or exertion; whilst the so-called movable kidney is so freely movable in any direction that it returns to its bed with as much ease as it quitted it. But there is another analogy with a dislocated bone. Like an ordinary dislocation it can be reduced by judicious management, or may in some rare instances remain unreduced, in which case a more or less complete atrophy takes place.

Case 1.—One of the most typical forms of this affection that I have ever come across was presented by G. P—, æt. 22, who was under my care in St. Bartholomew's Hospital in 1890. He was employed in a carpet warehouse in the City, chiefly in lifting heavy carpets. About

a year previously to coming under my care he had suffered at times from severe pains over the region of his left kidney, and had been most judiciously treated by Mr. Fletcher, of Charterhouse Square. Rest speedily caused an abatement of his pain, but having experienced an unusually severe attack in September, accompanied by hæmaturia, he had been obliged to lie up for several days, and very shortly after resuming work he was again prostrated, and obliged to seek medical aid.

At Mr. Fletcher's request he came under my care and was admitted into the Darker Ward, where he gave the following account of his last attack, which was then so fresh in his mind. He was lifting up a heavy carpet to put it on a shelf, which he almost succeeded in accomplishing, when all of a sudden a severe sickening pain seized him in his left loin, and he let the carpet drop upon the floor. For some few minutes he was almost unable to move, but managed to get home, though with difficulty. The pain became much more severe that night, and his side both in front and behind acutely tender. The next day he kept his bed; but the day following, when the symptoms were beginning to abate, he went to see Mr. Fletcher, who at once sent him on to me. He was admitted on the same day and put to bed. His side, in the region of the left kidney both behind and in front, particularly in the latter situation, was tender, and yielded a very tympanitic note to percussion.

A purgative was administered, and he was kept in a recumbent position. In a few days his pain entirely disappeared. When he was examined three days later there was no pain or tenderness whatever, nor was there any renal enlargement to be discovered. The urine which he had passed since admission was carefully examined, but beyond being of rather low specific gravity no abnormal condition was present. He remained in the hospital from October 6th until November 22nd, during most of which time he was up and about, making himself generally useful and enjoying excellent health. No

further attack ensued, and he was advised to go back to work, but was cautioned to come back at once should any tendency to pain return.

On December 15th he again presented himself, saying he had a slight pain which he was sure was the precursor of another attack, and such in a few hours it proved to be. Later on in the afternoon I made an examination of his abdomen, which had not yet begun to be very tender, and found the kidney distinctly enlarged, probably to treble its normal size, and whilst I was manipulating it it suddenly slipped from under my grasp, and a few hours later he passed a copious amount of clear urine of low specific gravity. He was anxious to go home, and did not return until the following day. He then felt perfectly well. During the next few days he was purposely made to exert himself; another slight attack came on, which was cut short by manipulation of the abdomen. He was advised to submit to an exploratory operation, with a view to ascertain the exact condition of affairs and carry out whatever operative treatment might be necessary. Before the day on which the operation was to be undertaken another attack came on, and whilst the kidney was enlarged and in a state of tenderness, the patient was placed under an anæsthetic, and an incision made parallel with the last rib behind. The kidney was found without any difficulty, and was at once seen to be considerably enlarged and tense. It was about the size of a large cocoa-nut, and was evidently filled with fluid. An incision was made into it, and the fluid spurted out from its interior, but no calculus could be detected. The walls were thinned out, and contained little if any secreting substance. During the process of exploring the interior of the cavity with the finger, and whilst the outflow of urine through the incision was prevented by the finger with which the kidney was being explored, the fluid began gradually to escape down the ureter, until the cyst was all but emptied. Whether this was due to the unkinking of the ureter or no could not be made out with

absolute certainty, but this seemed to be the most probable solution of the case. As the kidney was so completely disorganised it was removed, and the patient made an excellent recovery. At the present time, December, 1892, he is following his old employment, and has never suffered from any attacks of pain since the loss of his kidney.

Case 2.—This case illustrates very well the way in which some of these cases are produced, injury being in some instances the exciting cause.

W. E. P— was sent to me by Mr. Maund, an old house surgeon of mine, in September, 1892. He stated that in March of the same year he had been thrown out of a cart containing a load of wood which he was driving in Mashonaland. He was buried under the wood, and taken to a hospital some miles away, where he remained for seven weeks, suffering considerable pain in the abdomen from time to time. During the early part of his stay in hospital he passed blood with his water, but this soon passed off. As soon as he became well enough to be moved he made his way south to Capetown, and embarked for England.

In the course of his journey from Mashonaland southwards to Capetown he travelled partly by waggon, as he was not strong enough to ride, and he had several attacks of hæmaturia accompanied by severe pain, which he distinctly remembers to have felt in his left lumbar region.

When he started on the voyage he came across Mr. Maund, who was ship surgeon, and who witnessed two or three of his attacks on board ship, and has furnished me with a most accurate account of them. He had two or three of them on his voyage, and each of them came on in rough weather, and appeared to be distinctly traceable to slipping on deck when the ship lurched. As soon as this occurred he was seized on this occasion with a sharp pain in the left lumbar region. It radiated

down into his groin and thigh. His abdomen on the left side became very painful, and the pain gradually got worse, until it was so severe that morphia had to be employed to moderate it. He was violently sick, and sweated profusely, and passed some blood with his water. On one occasion his abdomen swelled to such an extent that he burst his flannel binder. Each attack usually lasted about seven or eight hours, and seemed almost suddenly to decrease in violence, after which it speedily disappeared, perhaps after the lapse of an hour or two. Its disappearance was always accompanied by the passage of a considerable amount of clear urine of a low specific gravity.

In addition to these severer attacks he had one or two threatenings which passed off apparently in less than an hour, and he has also had one or two slighter attacks since landing in England.

On September 6th, 1892, I saw him, and made a very complete examination of his abdomen. There was nothing at all abnormal to be discovered, except that just below the region of the left kidney there was a very slight amount of tenderness on deep pressure.

Having regard to the fact that all the recent attacks from which this patient had suffered had been most probably produced by the violent jolting which he had experienced in the Cape waggon, and to the rolling on board the steamer, I advised him not to be in any hurry to court operative interference, but at first to be content with moderate exercise, gradually increasing its amount and extent. This, I learned in November, he had done, and had had no further attacks of violent pain, though some discomfort still existed. One may reasonably hope that his kidney has again become fixed in position, and will not require any artificial aid to retain it there.

Case 3.—J. G—, æt. 39, was quite well up to her last confinement. Since that time she has complained of a good deal of pain in the region of her left kidney. The

pain is not constant, but it is seldom that many days elapse without an attack. The attacks last from three or four hours up to as much as eight or ten, but seldom longer. They sometimes come on without any apparent cause, but generally after exertion. The onset is invariably sudden, and accompanied by a severe pain which often shoots down into the groin and thigh. During the time which they last she is obliged to lie down, and is totally incapacitated from following her employment. Severe nausea and sometimes vomiting accompany the pain. She does not remember ever having passed blood with her water; but for the last six months micturition has often been painful, and her bladder is so irritable that she is often compelled to void her urine every quarter of an hour during the attacks. Menstruation quite regular since she ceased suckling.

Present condition.—She is a well-nourished woman, and with the exception of the attacks above referred to enjoys good health.

Shortly after coming into the West London Hospital one of her attacks came on one morning, shortly after she had passed a somewhat constipated motion, which was accompanied by a good deal of straining. About three hours after the onset of the pain her abdomen was examined, and a swelling was found about midway between the ribs and the crest of the ilium on the left side. It was rounded and painful to the touch. Handling gave rise to feeling of nausea. The whole of the abdomen on the affected side was more tender than natural, and there was an increased sense of resistance. She was kept perfectly quiet in bed, and as the pain was not very severe when she remained quite quiet no morphia was administered. Hot flannels, applied to the affected part, relieved her considerably, and about six hours after the attack came on it passed off almost as suddenly as it started.

The urine, which was passed once or twice an hour during the attack, was normal in appearance, highly acid, sp. gr. 1016, but contained no blood or albumen.

When she was seen two days later she was up and in her usual health. The abdomen was examined, and was discovered to be slightly more tender over the region of the left kidney than elsewhere. By bimanual examination the left kidney could be distinctly felt and moved from its proper place, but little or no pain was experienced during the examination. As there seemed to be no doubt that her symptoms were dependent on some mobility of the kidney an operation was proposed, consented to by the patient, and performed a few days later.

July 6th, 1892.—Under ether the kidney was exposed by an oblique lumbar incision, and was found without any difficulty. It was very movable, and slipped away from one's grasp, but the pressure of an assistant's hand on the front of the abdomen readily brought it up into the lumbar wound. After its fatty envelope had been cut through and partially removed, two silk sutures were passed deeply through the kidney substance and then fixed to the muscular structures in the lumbar region at the back of the abdomen.

The wound was closed by four deep sutures and some superficial ones, after which it was dressed with cyanide gauze, and the abdomen firmly bandaged.

The patient was rather collapsed after the operation, but soon rallied, and made an excellent and rapid recovery.

The urine was somewhat bloodstained after the operation for a day or two, and irritability of the bladder persisted for a few days longer.

The wound was dressed on the eighth day and the stitches removed, as it was all united. She remained in bed three weeks, after which she began to get up, at first for a few hours only. She left the hospital on August 4th, having had no attack of pain since the operation.

December 4th, 1892.—Came to show herself, and stated that though once or twice she had felt some aching pains in her back she had never experienced any of her old attacks of pain or of vesical irritability.

Case 4.—M. M—, æt. 23, was seen by Dr. Swindell, of Finchley, on December 2nd, 1889. She had been seized a few hours before he saw her with violent pain in the region of the left kidney, so violent that she called out with the agony. A few hours later I saw her in consultation with him. The pain was then slightly less, as she had had a morphia injection. She could not account in any way for her attack, unless it was due to stretching up to put some things on a shelf, which she had been doing just before her attack came on. When I saw her, about seven hours after her attack had begun, she was sweating profusely, her temperature was 100·6°, and she had vomited once or twice. The abdomen on the left side was exquisitely tender to the touch; it was somewhat distended and tympanitic, and she had passed water a considerable number of times. The water was clear, but contained a trace of albumen; sp. gr. 1010. The diagnosis was at the time doubtful, and the question of an attack of peritonitis due to some rupture of the gut passed through my mind. On the whole, however, the urinary irritability coupled with the presence of albumen seemed to point to the kidney as the source of the trouble, and I contented myself with prescribing rest in bed, morphia to allay pain, hot stupes to the abdomen, and a purgative. I did not see her again, as the attack passed off after a a few days. The next day some bloodstained urine was passed, and a few days later the patient was up and about again as usual. No calculus was discovered, though carefully searched for. There has been (Dr. Swindell writes me December 5th, 1892) no attack since.

Case 5.—J. E—, æt. 33 (servant), came under my care at the West London Hospital on December 4th, 1890, for severe pain in the right lumbar region. She had first noticed the pain, after a hard day's work, about eighteen months previously, and latterly her pains had become more severe. At times, as in the other cases referred to, her attacks were accompanied by nausea and vomiting.

An examination of the abdomen revealed a kidney on the right side which was more freely movable than it should be, and tender to touch. The urine was acid, sp. gr. 1014, and contained no albumen. Nephrorrhaphy was performed by the method previously described, the sutures being passed through the kidney substance. The patient made a good and rapid recovery, and when seen rather more than eighteen months later, said she had experienced none of her attacks of pain since leaving the hospital.

A very similar case to those just alluded to has been published by Guyon,[1] and is in several particulars so remarkable that no excuse need be made for introducing some of its details into the present account. Two points are especially interesting: in the first place the kidneys are distinctly stated to have been movable only to a slight extent; and in the second it presents an example of the acute and chronic variety of movable kidney occurring at different times in the same individual.

Madame X—, æt. 35, had enjoyed excellent health up to the age of twenty-nine. Married in 1879, she had four pregnancies, the first in 1881, the second in 1882, the third in 1884, and the fourth in 1887.

In February, 1881, about a month after her child was born, as she was stooping to pick up something from the floor, she suddenly experienced a severe pain in the region of her right kidney. During the first few hours the pain was endurable, but soon became rapidly augmented in intensity, lancinating, and violent. It seemed, said the patient, as if some one was sawing into her side between the crest of the ilium and her ribs. This was soon followed by vomiting, pallor, and an anxious countenance. Under the influence of a subcutaneous injection of morphia the severity of the attack passed off, and on

[1] 'Annales des Maladies des Organes génito-urinaires,' tome ix, p. 653: "Retention rénale aseptique intermittente (hydronéphrose). Guérison par malaxation et fixation orthopédique." The case is further alluded to by Arnould in his thesis, 'Contribution à l'étude de l'hydronéphrose,' Paris, 1891.

the following day, with the exception of a sensation of lassitude, Madame X— felt little the worse for her attack. For the next two or three months the patient experienced a good many similar attacks, which varied considerably both in their duration and severity. They usually lasted from four to ten hours, but readily yielded to morphia. The urine, which was frequently examined, never exhibited a trace of gravel or of blood.

During the next two or three years the attacks persisted with a varying, though apparently decreasing, intensity. Towards the end of 1884, *i. e.* about three and a half years after the first onset of the pain, her medical attendant discovered just at the end of one of her attacks a swelling in the region of the right kidney, which was slightly tender and slipped away from under his grasp. He at once suspected a movable kidney. On several subsequent occasions he encountered the same swelling, always exhibiting the same characters. It was movable to a *very slight* extent, and appeared to be situated deep down in the abdomen. During the latter months of 1885 the symptoms entirely disappeared, and did not return again till four years later, viz. in 1889. At first she complained of little but a general malaise and dragging sensation, but the attacks soon increased in violence, and became almost as severe as they were at first. The gastric symptoms were particularly severe, and she not only vomited frequently, but took a great disgust to her food. All the time her menstruation was perfectly regular and normal, and seemed to have no share in the production of her symptoms.

Her urine exhibited traces of albumen, but otherwise there was nothing abnormal in it.

The later history of the case is also very instructive. In 1889 she was advised to wear a belt containing an elastic ball, and so long as she kept her kidney in position she suffered but little inconvenience and no recurrence of the attacks of nausea and pain.

From a consideration of several of the above cases it

will be seen that not merely has one to deal with a displacement of the kidney, but that hydronephrosis may be present as well. The association of these two conditions has been noted by many authors; by Urag, 'Wien. med. Woch.,' 1856; by Wagner, 'Berlin. klin. Woch.,' 1881; by Landau, Sonn, Newman, Morris, and others; but it is only quite within the last few years that the interdependence of these two conditions has been mentioned, and even at the present time it has not obtained a general credence. In a paper on the connection between hydronephrosis and movable kidney ('Bull. de l'Acad. de Méd.,' February, 1889), Guyon calls attention to the curving of the ureter which he has observed in some cases of hydronephrosis, and points out that curvature implies lengthening, and that lengthening depends upon undue mobility of the kidney, a proposition which he still further proves by describing minutely the conditions which he found in operating on several cases.

It is not, however, my purpose in this present paper to insist so much on the relationship of these two conditions as to show that a dislocated kidney which is not freely movable gives rise to a condition of local congestion and temporary hydronephrosis, which is often associated with very severe pain, and by the other conditions which have been just detailed.

It is curious to observe how little attention, especially in England,[1] has up to this time been paid to this variety of hydronephrosis.

Roberts dismisses it in a few lines. Speaking of hydronephrosis,[2] he says, "The tumour is usually quite painless,

[1] Mr. Clement Lucas has drawn my attention to the fact that I have overlooked a paper of his ('Brit. Med. Journ.,' 1891, vol. i, p. 1343), in which he has insisted on the relationship of hydronephrosis to movable kidney. I am sorry this paper should have escaped my notice. It does not, however, appear to me, after reading it, that Mr. Lucas has laid sufficient stress on the acuter form of movable kidney to which I have applied the term acute renal dislocation.

[2] 'A Practical Treatise on Urinary Disease,' by Wm. Roberts, M.D., 4th edit., p. 551

and unaccompanied by any inconvenience except from its bulk. Occasionally, however, tenderness exists over it, and the action of the bowels is irregular. When the dilatation arises from the impaction of a calculus, symptoms of nephritic colic occur at the time when the impaction takes place, or from time to time thereafter if, as is most usual, some quantity of urine still continues to trickle past the calculus. Simple paroxysms are recorded in two instances where no calculus existed."

Morris gives the matter but scanty notice. He merely mentions, amongst the symptoms of hydronephrosis, "vomiting more or less frequent and severe, and sometimes preceded by a burning pain in the epigastrium." There is no special mention of paroxysmal attacks such as have just been recorded.

Nor is greater prominence given to the subject in Le Dentu's excellent and exhaustive treatise.[1] He says, "The onset of hydronephrosis is usually insidious. Although it is often produced by the incomplete obstruction of the ureter by a stone, attacks of nephritic colic rarely form a prominent feature in the histories of such patients. Frequently they only complain of a sensation of weight in the lumbar region, though sometimes painful sensations accompanied by *febrile reaction* are present;" and this is the more curious as painful attacks of hydronephrosis were described by Dietl[2] in 1864, and his account, together with Rollet's,[3] has been incorporated by Landau[4] in a work which has been admirably translated by Dr. Champneys, and forms a part of vol. cx of the 'New Sydenham Society's Transactions.'

More recently still Arnould, in an elaborate treatise on hydronephrosis which has already been referred to,

[1] 'Affections chirurgicales des Reins, des Uretères, et des Capsules surrénales,' par A. le Dentu, Paris, 1889, p. 440.

[2] 'Wandernde Nieren und deren Einklemmung," 'Wien. med. Woch.,' 1864, Nos. 36—38.

[3] 'Pathologie et Therapie der beweglichen Niere,' Erlangen, 1866.

[4] 'Die Wanderniere der Frauen,' Berlin, 1881.

has more fully explained the causes which lead up to hydronephrosis, and amongst them has included movable kidney.

One may fairly ask how it is that such obvious symptoms have so far failed to obtain due recognition. It cannot be that they have not been observed. It would seem more reasonable to conclude that the pains which accompany these attacks were formerly ascribed to the passage of a calculus, and that surgical interference has demonstrated not only the absence of a calculus but the presence of a movable kidney, and has further shown that such cases are especially favorable for nephrorrhaphy, since the probability of permanent cure is almost absolutely certain.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series. vol. v. p. 90.)

ON THE

REMOVAL OF A "PRESSURE-POUCH" OF THE ŒSOPHAGUS.

BY

HENRY T. BUTLIN, F.R.C.S.,

SURGEON TO ST. BARTHOLOMEW'S HOSPITAL.

Received December 14th, 1892—Read April 25th, 1893.

Towards the close of the year 1890 I was consulted by a gentleman forty-seven years of age, on account of the symptoms of an œsophageal pouch, namely, the return of undigested particles of food some time after they had been taken, and the presence of an ill-defined swelling on the left side of the neck, which became larger after a meal, and out of which gas and particles of food could be pressed. Captain X— dated his trouble from an attack of fever on the west coast of Africa some four years previously. He had suffered from similar fever on several occasions, but this particular attack had been accompanied by a violent cough which had, he believed, "wrenched his gullet."

He had not any signs of stricture of the œsophagus, and his general health was very good.

I had read of similar cases, but had never seen one; and I did not know what had been done for the cure of the disease. I therefore told the patient that I feared that nothing less than an operation would suffice to cure

him; that I did not know whether the disease had ever been treated by operation; and that I would look the matter up, and let him know in the course of a few days what could be done for him.

A study of text-books and papers showed me that pressure-pouches are extremely rare, and that the outlook of the patient is very gloomy. Although more than one author suggested the desirability of attempting to treat the condition by operation, I could not find a single instance in which the suggestion had been carried into effect. I therefore wrote to Captain X—, and told him I could not find any account of an operation in such a case as his; but if he grew worse and were disposed to try the chances of an operation, I should be prepared to perform it for him, after consideration of the best means of exposing the pouch and a consultation with some other surgeon.

In the spring of 1892 Sir William Dalby, who was connected by marriage with the patient, wrote to tell me Captain X— had grown much worse; that he was now unable to get down his food without difficulty, and could not venture to dine in company; and that his trouble was producing an ill effect on his mind and body. Sir William Dalby talked over the question of operation with me, and I told him what I had previously told the patient. Nor could I add that any operation had been practised during the eighteen months which had elapsed since I had first been consulted on the case.

I doubt whether Captain X— would have submitted to an operation even now, but it so happened that I learned from Mr. Edgar Willett that a case of œsophageal pouch had been operated on by Professor von Bergmann with success. The case is published in full in the 'Archiv f. klin. Chirur.' (Bd. xliii, Hft. 1).[1]

[1] After this paper had been sent in I learned from Dr. Keser that Professor Kocher, of Bern, has published an account of a similar operation; it may be found in the 'Correspond. Blatt. f. Schweisser Aerzte,' xxii, No. 8, April 15th, 1892. Professor Kocher has operated in two cases, and in both with success.

As soon as I had communicated this circumstance to Sir W. Dalby, he urged Captain X— to place himself under my care, and to undergo an operation if I advised it.

It was arranged that the operation should take place on the 14th of June, and in the meantime Mr. T. Smith and Sir James Paget saw the patient with me in consultation. Both surgeons agreed that the case was as good a one for operation as could well be found, but Mr. Smith was disposed to recommend that the daily passage of a tube should be tried over a long period of time before an operation was resorted to. The friends of the patient also consulted Mr. Treves on the general question of the advisability of operation in such a case, and received great encouragement from him. The patient himself, a very courageous man, was determined to press for operation even if the opinions had not been so favorable to it in his own case.

June 14th (operation).—Sir James Paget and Sir William Dalby were present, while Mr. Mills gave chloroform, and Mr. Edgar Willett assisted me.

A long incision from just above the sternum, along the border of the sterno-mastoid, up to the level of the hyoid bone afforded ample space. The omo-hyoid muscle was divided; the superior thyroid artery exposed, seized with clamps, tied, and divided; the carotid sheath laid bare and drawn away from the middle line. The larynx was rotated on its long axis by drawing forward the left ala of the thyroid cartilage with blunt hooks, so that the pharynx was separated from its bed and its posterior surface presented towards the left side. The pouch was found without difficulty projecting to the left side behind the junction of the pharynx and œsophagus. It was freed from the surrounding connective tissue, and before removing it, in order to be quite sure that it was the pouch, a long probe was passed into it from the mouth. At the point where it joined the œsophagus its neck was very slightly constricted, as if by the thick muscular fibre round it, while the body of the sac seemed to be bare of

muscle. It was cut away from above downwards, and the margins of the cut wound were fastened together with eight sutures of fine silk as the sac was cut away. There were no bleeding vessels. The wound was filled from the bottom with iodoform gauze; only the upper and lower ends were brought together with sutures. This was done in order to ensure the free escape of any liquid which might ooze through from the œsophagus. A long india-rubber tube was passed into the stomach for the purpose of feeding, and the end of the tube was fastened to one of the teeth.

The operation was performed throughout on the same lines as those adopted by Professor von Bergmann, to whose excellent description I am much indebted.

The feeding tube caused so much irritation and sickness that it was removed in the evening; for I thought the patient might as well run the risk of taking good food down the œsophagus as of bringing up half-digested food from the stomach. He was fed throughout by the mouth.

16th.—The iodoform gauze was removed, and some semi-purulent liquid was let out.

17th.—A little liquid food issued from the wound. As it was exceedingly difficult to pass gauze to the bottom of the deep and sinuous passage, a piece of india-rubber tubing was introduced instead.

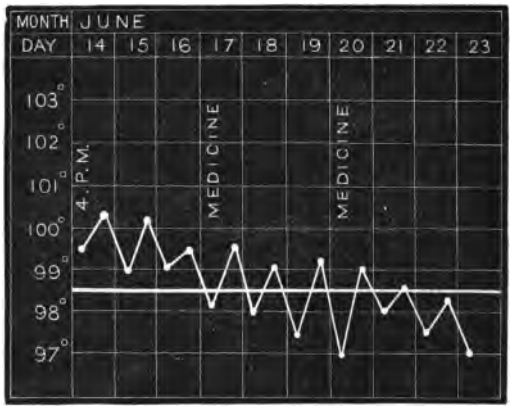
20th.—The slight doubt which had existed of whether the discharge contained food was dispelled by the discovery that water could be syringed through the external wound into the œsophagus. The upper and lower parts of the wound were healing rapidly.

27th.—It was no longer possible to syringe water into the œsophagus, and no food could be discovered in the small quantity of healthy pus which the wound discharged. The patient was so well that on the previous day he had driven down to Rutland Gate. The temperature had never risen above 100° from the time of the operation.

There is nothing more to relate of the progress of the case further than that the wound was completely healed

by the middle of July. I last saw Captain X— in November, 1892, when his scar was perfectly sound and good; he could swallow all kinds of food without discomfort, and was only conscious of having undergone a surgical operation by the slight numbness of the skin in front of the long scar.[1]

The comparative rarity of pressure-pouches of the œsophagus would itself be a sufficient excuse for bringing this case before the Society, in order to place on record



one more case. I do not know that it adds much to our knowledge of the causation of the condition. The pouch was about an inch and three quarters long at the time of removal, and empty. It was flattened from side to side, and the opening into the œsophagus was almost like a slit, and about an inch in length. There was no obvious layer of muscle covering it, although muscular fibre was thick about its neck. And Mr. Edgar Willett, who carefully examined it before it was set up for the museum of St. Bartholomew's Hospital, could not find any layer of muscle. The elasticity of the pouch was extraordinary; it could easily be distended to two or three times its natural size. I did not discover any cord attached to its free extremity, such as is sometimes attached to the base

[1] I saw him again in June, 1893: condition the same.

of a dermoid cyst. The absence of these things favours to some extent the theory of the origin of the pouch in pressure. And so does the occurrence of the first symptoms in all cases in adult life, and the much greater liability of males to the disease. Notwithstanding these objections, I cannot but feel strongly in favour of the view that such pouches have their origin in defective development. Their constant occurrence at the same part of the alimentary canal, and where it is supported by the vertebræ behind; their shape in this and many similar cases; their tendency always to pass out towards the left side; the slit-like opening through which they communicate with the alimentary canal; the very great rarity of their occurrence, in spite of the careless habits of eating of so many people, the resemblance of this pouch to some of the Meckel's diverticula of the intestine, were very marked; and although most of the Meckel's diverticula are covered throughout with muscular fibre, they are not all so to the free or distal extremity.

I had not originally intended to describe the position and symptoms of the pressure-pouch. But the published accounts of cases which have appeared within the last few years in surgical literature show that the diagnosis has not been made in some of these cases, and that the symptoms of the condition are not so well understood as they should be. The true pressure-pouch is invariably, or almost invariably, situated at the junction of the pharynx with the œsophagus, and it projects almost invariably at the back of the œsophagus, between the gullet and the spine. As it enlarges it bulges on one or both sides of the neck, below the level of the cricoid cartilage; if the bulging is on one side, it is almost always on the left side.

The early symptoms are the return of undigested particles of food some time, it may be many hours, after a meal has been taken. The return of food is sometimes associated with severe fits of coughing. After some time a larger quantity of food is returned, and the patient

becomes aware of some difficulty in swallowing, especially solid food. If a sound is passed, it can be introduced without difficulty, provided it does not enter the pouch, and it is thus clear that there is no stricture of the œsophagus.

At a later period swelling can be distinctly felt on one side or on both sides of the neck, and the patient often finds it possible to empty the pouch by pressure on the soft swelling. By these means the contents of the pouch may be squeezed into the œsophagus, and may pass down into the stomach. The swelling of the neck may be actually visible when the patient drinks a draught of water; it was so in the patient whose case is narrated in this paper. As the disease advances, and the pouch grows larger, the difficulty of swallowing food becomes greater, the bulging of the neck becomes more marked, and the passage of a sound is much more difficult, on account of its tendency to enter the pouch.

The operation proved much more easy than I had anticipated; partly, no doubt, because I had the advantage of having carefully studied von Bergmann's excellent description of his own operation, in which the removal of the pouch was made more difficult by the presence of considerable enlargement of the thyroid gland, the left lobe of which had to be turned aside before the parts could be exposed. Von Bergmann's suggestions of a very free incision and exposure of the deeper parts, and of rolling the larynx over on its long axis, proved particularly valuable; and I was really surprised at the ease with which the sac was discovered, freed, and removed.

Of the danger of such operations it is too soon to attempt to form a just opinion. Œsophagotomy is generally regarded as a dangerous operation; but I believe this is due rather to the conditions in which it is usually performed than to any special danger in the operation itself. In many of the fatal cases the operation had been performed for the removal of an impacted body, and death ensued from phlegmonous inflammation of the tissues around the œsophagus—an accident which is

attributable to the foreign body far more often than to the operation. And when the operation has been performed for stricture, the condition of the patient has usually left much to be desired. In cases such as that we are discussing, with a patient in good condition, with proper previous preparation, and with provision for the escape of liquids from the deep parts of the wound, I cannot but think that the operation will not prove dangerous. It is satisfactory to note that, although the stitches in the wall of the œsophagus did not suffice to prevent the passage of fluids through the wound two or three days after the operation, they were so far satisfactory that only a very small quantity passed during a few days. It is almost certain that part of the wound in the œsophagus was kept closed by them, and that they hastened the complete closure of the whole wound.

I can scarcely hope to perform this operation again, for the affection is so rare that it falls to the lot of but few surgeons to meet with two of these cases in the course of a long surgical life. But I trust the relation of this case may influence other surgeons to undertake the operation whenever the circumstances appear favorable. It is fully justified by the intractability of the affection to palliative treatment, and if not by the fatality, yet surely by the distress occasioned by the pouch. An analysis by Zenker and Ziemssen of twenty-seven cases taken from surgical literature, in which the diagnosis of the affection was confirmed by autopsy, shows that thirteen of the patients died of the results of the pouch; eight of them died of other diseases; while the cause of death of the remaining six was not stated, and the manner of death has been in most cases by slow starvation.

APPENDIX.

Those persons who wish to study a complete account of pressure-pouches or diverticula of the œsophagus will find excellent papers in the 'Handbuch der speciellen

Pathologie und Therapie,' vii, H. 1., 1874—1877, Zenker u. v. Ziemssen; 'Deutsche Chirurgie,' Lief. 35, 1880, Koenig; 'Diseases of the Throat and Nose,' vol. ii, 1884, Morell Mackenzie; 'Langenbeck's Archiv,' Bd. xliii, 1892, von Bergmann. In these works references will be found to other papers and works bearing on the subject.

Two cases have been lately published, one in the 'Lancet,' 1891, vol. i, January 3rd, by Mr. Walter Whitehead; the other in the Pathological Society's 'Transactions,' vol. xlii, page 82, 1891, by Mr. Chavasse. Both patients died. Gastrostomy was performed in both cases for the relief of the symptoms. In Mr. Chavasse's case the operation was delayed until the man had been too far reduced by starvation to profit by it. In Mr. Whitehead's case the woman died six years after gastrostomy had been successfully performed. She grew weary of feeding through the stomach opening, and thought her gullet trouble was cured. For a while she fed through the mouth, and the gastrostomy wound closed. Gradually, feeding through the mouth became more difficult, until she died of anæmia and exhaustion at the age of sixty-two. The true nature of the disease does not appear to have been ascertained until after her death. The first symptoms had been noticed at the age of forty-eight years.

Pressure-pouches of the Œsophagus in London Anatomical Museums, February, 1893.

Royal College of Surgeons.—No. 2291. An excellent specimen taken from the body of a bishop who died at the age of ninety years. The pouch is in the usual position at the back of the alimentary canal. It is about 2 inches long, said not to be covered by muscle; it has a wide orifice. Almost all the food used finally to pass into it, and was pressed out after each meal into the œsophageal canal. The cause of the patient's death is not stated, but it does not appear to have been caused by the pouch.

No. 2291 D. Mr. Chavasse's case ('Path. Trans.,' xlii, p. 82, 1891).

St. Bartholomew's.—The only specimen is the pouch exhibited with this paper.

Guy's.—No. 3528. A small specimen consisting of a portion of the pharynx and œsophagus, with a pouch about 1½ inches long, with an opening into the alimentary canal, circular in shape, about an inch in diameter. The diameter of the sac is rather more than an inch. As there are none of the adjacent parts in the specimen, and only a small portion of the wall of the gullet, it is scarcely possible to be sure whether the pouch projects from the back or side of the alimentary canal. There is no history of the case in the catalogue, and no reference to any note elsewhere. The specimen is probably very old.

St. Thomas's.—No. 900. A very interesting specimen showing a pouch about 3 inches long, opening in a very unusual position on the left side instead of at the back of the alimentary canal, but at the ordinary level near the junction of the pharynx with the œsophagus. The opening into the sac is circular, and about an inch in diameter.

St. George's.—No. 15 A, or IX, 14. Dr. Ogle's case, published in the 'Pathological Transactions,' 1866, p. 141.

University.—No specimen.

St. Mary's.—No specimen.

Charing Cross.—No specimen.

Middlesex.—No specimen.

Westminster.—No specimen.

King's College.—No specimen.

London.—Through the kindness of Mr. Treves I learn that this museum does not contain any specimen (letter, February 23rd, 1893).

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. v, p. 101.)

DESCRIPTION OF PLATE IV.

On the Removal of a "Pressure-pouch" of the Œsophagus (HENRY T. BUTLIN, F.R.C.S.).

FIG. 1.—Life-size drawing of the pouch removed by the author. A portion of the outer coat is stripped down on one side.

FIG. 2.—A side view of the specimen in Mr. Chavasse's case, to which reference is made in the Appendix. Slightly reduced in size. Anatomical Museum of the Royal College of Surgeons, No. 2291D. ('Path. Trans.,' vol. xlii, p. 82, 1891.)

FIG. 3.—Back view of specimen in the Museum of the Royal College of Surgeons, No. 2291. The posterior wall of the pharynx has been laid open, and the pouch lies over to one side. See Appendix.

Fig. 1.

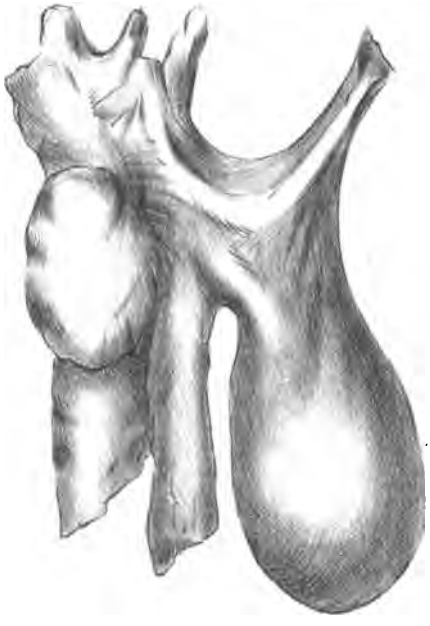


Fig. 2.

Fig. 3.

Danielsson & Co, lith.

DESCRIPTION OF PLATE V.

On the Removal of a "Pressure-pouch" of the Œsophagus (HENRY T. BUTLIN, F.R.C.S.).

FIG. 4.—Sketch of a specimen in the Museum of St. Thomas's Hospital. The pharynx and œsophagus have been laid open at the back. The opening of the pouch is quite to one side, which is very unusual in such cases. The pouch is not known to have produced any serious symptoms during life.

FIG. 5.—Sketch of an old specimen in the Museum of Guy's Hospital. There is no detailed description of the specimen in the catalogue, nor is there any account of the circumstances under which it was found.

FIG. 6.—Reproduction, a little diminished in size, of the drawing of the specimen (No. 15A, or IX, 14) from Dr. Ogle's case in the Museum of St. George's Hospital, published in the 'Path. Trans.,' 1866, p. 141. The opening of the pouch down the middle gives the impression that it has a larger mouth than is really the case. A study of the specimen corrects this impression.

The author is indebted to the authorities of the various museums for kind permission to use these specimens.

Fig 4.

Fig 5.

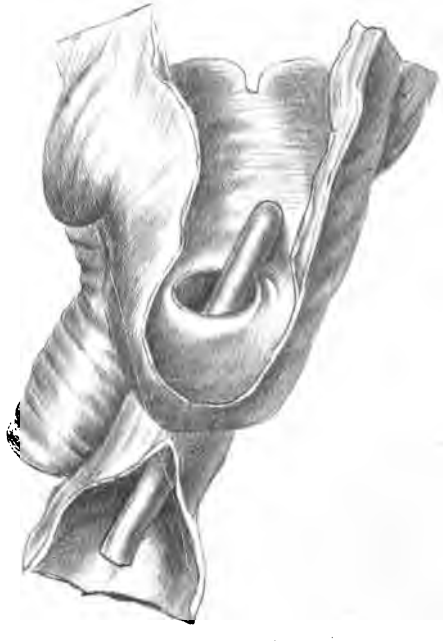


Fig 6.

Danielsson & Co. Lith.

ON THE

INFLUENCE OF VARIOUS DIETS UPON THE COMPOSITION OF THE URINE

AND

THE GENERAL CONDITION OF PATIENTS SUFFERING FROM CHRONIC BRIGHT'S DISEASE.

BY

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Received February 9th—Read April 25th, 1893.

During the last few years much has been said and written about the treatment of chronic parenchymatous nephritis and chronic interstitial nephritis. The discussion has turned mainly on two points—drugs and diet. Iodide of potassium, lactate of strontium, fuchsin, tannin, benzoic acid, nitric acid, extract of secale cornutum, bromo-benzol, and acetate of lead have all been tried, and no one of these has gained any permanent hold upon the good opinion of a large number of the profession; perhaps the most popular is iodide of potassium. From my own experience I think that lactate of strontium will sometimes diminish the albuminuria, but I have been unable to detect that the condition of the patient is in any

other respect altered by taking it. However, I do not propose this evening to deal with drugs, but to confine myself entirely to the question of diet in chronic Bright's disease.

I might be the whole evening quoting references to this subject, so I shall have to content myself with giving only the more important. The matter was discussed at the ninth Congress of Medicine[1] held at Vienna in 1890. It was introduced by Senator. He did not say much about chronic interstitial nephritis, but he strongly recommended milk diet for the chronic parenchymatous form, and if milk disagreed he advised buttermilk, koumis, or kef; or if these could not be obtained, fish or fowl.

At the tenth International Medical Congress held at Berlin in 1890[2] the subject was discussed at great length. Lépine introduced it, and said he thought milk was the ideal diet, for it replaces the albumen that the patient loses, all its nitrogen is absorbed and used, it does not irritate the kidneys, it is diuretic, and it does not contain any toxic substances, but aids their elimination. He, however, admitted that patients quickly get disgusted with milk, and therefore he also allows vegetables and farinaceous foods.

Grainger Stewart spoke next, and stated that an abundant diet of ordinary food increased the urea and albumen passed, a purely milk diet increased the flow of urine, diminished the discharge of albumen, and in some cases increased that of urea. The best results were obtained by a diet composed largely of milk, but also in part of bread, potatoes, sugar, and butter. Sometimes the milk diet manifestly disagreed with the patient, increased his renal symptoms, and at the same time rendered him uncomfortable and dyspeptic. Milk diet he considered to be especially useful in cases of inflammation of the tubules, and as the case approaches the chronic interstitial variety, so in proportion is milk

[1] 'Centralblatt f. klin. Med.,' 1890.

[2] 'Verhandlungen des X Intern. Med. Congresses,' Bd. ii, Abtheilung 5.

less valuable. Fuller details of Grainger Stewart's opinions will be found in his work on albuminuria.[1] Three cases of parenchymatous nephritis (p. 203) were carefully tested with different diets. In the first ordinary diet, milk diet, and low diet brought out essentially the same result, both in respect to urea and albumen. In the second case it was somewhat difficult to judge the results, for the patient was steadily recovering, but on the whole milk and low diets were better than ordinary diets, and low diet was the best of all. In the third case the diet made no difference to the amount of urine nor to its specific gravity, and it hardly affected the urea, but the albumen diminished under milk, and continued to do so on low diet. The author believes that these experiments do not warrant our arriving at any general conclusion, although they to some extent support the view that milk or low diet is better suited to cases of parenchymatous nephritis than ordinary or ample diet. One case in which an acute attack of Bright's disease supervened upon the chronic form was dieted with different diets, but no corresponding effect was discernible upon the quantity of urine or urea, and the albuminuria was certainly not diminished by the milk. And, lastly, the conclusion was reached that in the cirrhotic form of Bright's disease the diet is of less importance than in tubal inflammation.

Drs. Sparks and Mitchell Bruce[2] have read an important paper on the effect of diet, rest, exercise, &c., on a case of chronic nephritis. They came to the conclusion that the amount of albumen was diminished by an absolute milk diet, which, however, produced a sensation of unsatisfied hunger and sinking, and by an absolute non-nitrogenous diet, and that the milk diet caused a much diminished secretion of urea. The exact form of chronic nephritis from which this patient was suffering was perhaps doubtful.

[1] 'Lectures on Important Symptoms. II. Albuminuria.'

[2] 'Trans. Roy. Med.-Chir. Soc.,' vol. lxii.

Rosenstein[1] is much more liberal in his dietetics than most authors, for, provided that the digestive functions are good, he allows milk, eggs, meat, green vegetables, and good red Bordeaux to patients with chronic nephritis.

Osler[2] advises for acute Bright's disease a diet consisting of milk, buttermilk, gruel, barley water, chicken broth, beef-tea; and considers that, if possible, the patient should be kept on a strictly milk diet. For chronic parenchymatous nephritis he recommends a similar dietary, and he thinks that no particular diet is necessary for patients with chronic interstitial nephritis so long as whatever they have is taken in moderation.

Fenwick[3] states that patients with chronic Bright's disease should eat but little meat and plenty of vegetables and fruit, but he quotes Niemeyer as strongly recommending a milk diet. With regard to acute Bright's disease, he points out that the majority of French writers advise any light diet, but in Germany a purely or almost purely milk diet is given.

Sir George Johnson[4] says that in cases of acute albuminuria, if the albumen has disappeared on milk diet, the addition of solid food will cause it to reappear; and with regard to more chronic cases he advises milk diet, and relates the case of a man who lived on milk for a long time with great benefit.

Ralfe,[5] too, mentions a case of chronic nephritis in which much good accrued from a milk diet.

Sir William Roberts[6] simply says that milk agrees well with patients suffering from chronic Bright's disease, and that they may partake freely of it. For the acute disorder he advises light farinaceous diet.

[1] 'Nierenkrankheiten,' 3te Auflage, S. 343, 1886.
[2] 'Principles and Practice of Medicine,' 1892.
[3] 'Outlines of Medical Treatment,' 1891.
[4] 'Medical Lectures and Essays.'
[5] 'Diseases of the Kidney.'
[6] 'Diseases of the Kidney,' 4th edit. Edited by Maguire.

Dickinson[1] would restrict patients to the smallest amount of animal food on which they will thrive.

Labadie-Lagrave[2] does not very strongly advise milk, but thinks it is most useful for patients suffering from a large white kidney.

Carter,[3] in his lectures on uræmia, states that the urine of animals fed on milk was less poisonous than that of those fed on other diets, and he recommends a milk diet in Bright's disease.

Zasiadko[4] made a series of experiments from which he concluded that in chronic Bright's disease, on vegetable foods, the albumen decreased, arterial tension sank, the dropsy increased, the pulse became slower and weaker, the appetite was lost, and the general state of the patient was worse; but on animal diet the albumen rose in quantity, the pulse was stronger and its tension was higher, the œdema diminished, the amount of the urine, its proportion of solids, and its specific gravity all increased, and the general state of the patient improved. With a mixed diet intermediate results were obtained. Therefore for interstitial nephritis he advises chiefly animal food, but for the parenchymatous form he recommends vegetable diet.

The subject was discussed at a recent meeting of the Académie des Sciences. Dujardin-Beaumetz said that the object of treatment should not be so much to affect the albuminuria as to diminish the risk of poisoning by toxic substances; he therefore suggested that all food should be fresh—apparently assuming that the toxines which cause uræmia are the same as those which develop in decomposing food. Consequently he forbids fish, game, oysters, and cheese. He regards milk as very valuable, but advises that it should be sterilised, and says meat should be thoroughly cooked. He never saw albuminuria increased by eggs.

[1] 'Diseases of the Kidney.'

[2] 'Urologie clinique et maladies des reins,' 1888.

[3] 'British Med. Journal,' Sept. 1st, 1888.

[4] 'British Med. Journal, Epitome,' Nov. 8th, 1890.

I might quote many other authors, but I think I have referred to sufficient to show that they are by no means unanimous as to the best diet to give patients suffering from chronic Bright's disease. For this reason, and also because many have used too few cases for the results to be conclusive, and others have relied largely on *a priori* considerations, it seemed to me worth while to carefully analyse the urine and note the symptoms in a series of cases. We know so little that is certain about the cause of the serious symptoms of Bright's disease that it is only by actual trial that we can find out whether any particular diet is valuable.

Milk is very frequently recommended, and it has been claimed that it will do good for the following reasons:

1. It is said to diminish the amount of albumen. In the first place, the figures I shall give later on will show that this is very far from being always true, at any rate in chronic interstitial nephritis; and some of Grainger Stewart's cases point in the same direction, for on ordinary, large, milk, or low diet the albuminuria remained unchanged, and even partaking abundantly of eggs did not increase it. Such experiences as these mean either that the reported cases in which it is said that the albuminuria was diminished by a milk diet belong to a different category, or else that they were improving in this respect at the time that the milk diet was started.

Then, secondly, even if milk did decrease the albuminuria, we have in many cases no proof that this is of any benefit. Patients who are very ill may lose only one or two grammes of albumen a day in the urine. This cannot of itself be of much importance; the absorption from the intestine of a very little more albuminous material would, if not lost in some excretion, quickly make up the deficiency. Then, again, people who suffer from cyclic albuminuria are often none the worse for it, nor does the loss of albumen in other ways, as by epistaxis, or by means of a discharge of pus, produce any symptoms comparable to those of Bright's disease.

Indeed, the majority of persons who pass albumen in their urine have not got Bright's disease at all, but are suffering from heart disease, pyuria, specific fevers, &c. Lastly, albuminuria is probably to a large extent only a local sign indicating disease of the secreting renal epithelium, and not a general blood condition. All these considerations indicate that even in cases of Bright's disease in which the loss of albumen is considerable, this of itself is not really quite so important as is usually thought. Surely if it were, restricting the diet to food containing less albumen than ordinary diet ought to be harmful to the patient, for not only would he be losing more albumen than in health, but he would be taking in less, and I shall presently show that—if we may assume that when more proteid is taken by persons suffering from Bright's disease more is absorbed—even when full diet increases albuminuria, the increased amount of proteid taken very much more than compensates for the increased albumen passed in the urine, and in some cases of Bright's disease the albuminuria is even less on full than on milk or farinaceous diets. No doubt the restriction of the saccharine food in diabetes would by analogy lead us to restrict the albuminous food in albuminuria, but analogical reasoning of this sort is most fallacious. I feel quite sure that the significance of albuminuria in Bright's disease is over-estimated, and that treatment directed immediately to it is unsound, especially when, as in the case of milk diet, although it frequently fails to diminish the amount of albumen passed, it often sets up loathing and disgust.

2. Some authors urge that milk is easily digested and absorbed. Often this is directly contrary to fact. Many writers have noticed, and I have often observed, that patients who have chronic Bright's disease and are fed solely upon milk often suffer from indigestion, and, as just mentioned, they get to positively loathe the milk, which also has the additional disadvantage that it leads to constipation.

3. It is stated that milk does not irritate the kidneys. But before this statement can have any value it must be

shown that ordinary food does irritate them, and this has not yet been done. It is curious that if milk is such a particularly bland and unirritating substance to the kidneys, these organs should in young children be so frequently streaked with urates.

4. Milk is often said to be diuretic. For instance, the patient whose case is related by Drs. Sparks and Mitchell Bruce passed more urine on a milk diet than on ordinary diet together with a large amount of water; but as he passed more urine in this combination than on ordinary diet alone, it is highly probable that the diuretic effect of the milk was in part due to the quantity of water in it. Some of the cases I shall relate show that milk is usually, but not invariably, diuretic. Of Grainger Stewart's cases milk did not act as a diuretic in those with parenchymatous nephritis, nor did it in the chronic cases. Thus we see that milk is neither a powerful nor a certain diuretic; and we must remember that even if it were, it is doubtful whether it would be a good thing to give diuretics in Bright's disease.

(5) Lépine has urged that milk is beneficial for Bright's disease because it contains no toxic substances. But, for all we know, it may contain poisonous substances, which although excreted in health are retained in the blood in Bright's disease. His supposition that it aids the elimination of toxic substances in Bright's disease is without a shadow of proof. It seems to me futile to argue in this way about the toxine of Bright's disease when we do not know what it is.

The view that a diet containing very little proteid is beneficial for Bright's disease is probably due to a lingering survival of the false belief that the urea which is not excreted in Bright's disease is the cause of the symptoms of uræmia.

We thus see that all the *a priori* considerations which have been put forward as reasons for giving milk in chronic Bright's disease have in themselves no value, and similar objections could be urged against the reasons

which have led to the belief that other particular diets would be beneficial. Therefore all that is left to us is to examine cases, and I propose now to analyse the ten cases, all under my own care, which form the basis of this paper. The following is the composition of the diets employed.

Milk diet consists of milk 3 pints a day.

Farinaceous diet consists of bread 12 oz., butter 1 oz., milk 2 pints, beef tea 1 pint a day. (The beef tea is often omitted.)

Full diet consists of bread 12 oz., butter 1 oz. milk ½ pint, potatoes ½ lb., meat 6 oz. a day. Rice pudding made with milk ½ lb. is allowed on alternate days; when this is not given the patient has ½ pint of mutton broth. The patients whose cases are narrated in this paper nearly always had rather over a pint of milk instead of the above half-pint. Tea and sugar are allowed with both farinaceous and full diets. The amount of milk taken with the tea is small, and as it is the same in both diets it may in contrasting them be neglected.

The following table shows the amount of proteid in each diet:

	Proteid.
Milk Diet.	
Milk 3 pts. = 61·5 oz. (milk contains 4 % proteid) =	2·46 oz.
Farinaceous Diet.	
Bread 12 oz. (containing 8% proteid) ... ... =	0·96 oz.
Milk 41 oz. (containing 4% proteid) ... ... =	1·64 oz.
	2·60 oz.
Full Diet.	
Bread 12 oz. (containing 8% proteid) ... ... =	0·96 oz.
Meat 6 oz. (containing 18% proteid) ... ... =	1·08 oz.
Milk rather over 1 pt. (containing 4% proteid) ... =	1·0 oz.
Rice pudding with milk, ½ lb. (about 5·5% proteid) =	0·44 oz.
	3·48 oz.

Milk diet contains therefore 1076 grains of proteid.
Farinaceous diet „ 1137 „ „
Full[1] diet „ 1522 „ „

When any of the patients showed signs of uræmia they

[1] On the days when rice pudding is not given there will not be quite so much proteid taken.

were treated with subcutaneous injections of nitrate of pilocarpine, vapour-baths, and compound jalap powder or compound elaterium powder. Eight were instances of chronic interstitial nephritis. Two were examples of chronic parenchymatous nephritis; it is possible that these two may have had some lardaceous disease of their kidneys.

We may analyse the results under the following heads: —(1) The quantity of urine passed. (2) Its specific gravity. (3) The amount of albumen. (4) The amount of urea. (5) The general condition of the patient.

I. The Quantity of Urine passed.

(A) *Eight Cases of Interstitial Nephritis.*

Milk diet.—Cases 2, 3, 6, and 7.

Case 2 passed 42 oz. a day on milk diet, against 40 on full, and 37 on full with chop and egg.

Case 3 passed 55 oz. a day on milk diet, against 33 on farinaceous, 35 on farinaceous with chicken, and 33 on farinaceous with chicken and fish.

Case 6 passed 88 oz. a day on milk diet against 100 on farinaceous, and 52 on full.

Case 7 passed 49 oz. a day on milk diet, against 42 on full, and 40 on full with an egg.

Result.—In three out of four cases more urine (viz. about 3 or 4 oz., about 20 oz., about 8 oz.) was passed daily on a milk diet than on other diets; in the remaining case 12 oz. a day less were passed upon a milk diet than upon farinaceous. But Cases 2 and 3 were fatal, and perhaps the lessened quantity of urine on other diets was due to the fact that the patients were nearing their end when taking these diets. On the whole it appears that milk is sometimes but not always a mild diuretic in chronic interstitial nephritis. There is, however, but little opportunity in these cases for contrasting it with farinaceous diet.

Farinaceous diet.—Cases 1, 3, 4 (two trials), 5, 6, and 8.

Case 1 passed 75 oz. a day on farinaceous diet, against 57 oz. on full.

Case 3 passed 33 oz. a day on farinaceous diet, against 55 oz. on milk, 35 oz. on farinaceous with chicken, and 33 oz. on farinaceous with chicken and fish.

Case 4 (first trial) passed 47 oz. a day on farinaceous diet, against 43 oz. previously on farinaceous with fish, and 39 oz. previously on full, and 50 oz. subsequently on farinaceous with fish, and 29 oz. on farinaceous with chicken.

Case 4 (second trial) passed 27 oz. a day on farinaceous diet, against 29 oz. previously on farinaceous with chicken, 21 oz. subsequently on full, and 20 oz. on farinaceous with fish.

Case 5 passed 36 oz. a day on farinaceous diet, against 20 oz. previously on farinaceous with milk, 29 oz. on farinaceous with fish, and 55 oz. subsequently on full.

Case 6 passed 100 oz. a day on farinaceous diet, against 88 oz. previously on milk and 52 oz. subsequently on full.

Case 8 passed 36 oz. a day on farinaceous diet, against 33 oz. subsequently on farinaceous with fish.

Result.—Contrast with milk. In one case less urine was passed on farinaceous diet (viz. 20 oz.), and in one case more (viz. 12 oz.).

Contrast with full diet. In four cases more urine was passed on farinaceous diet (viz. 18 oz., 8 oz., 6 oz. 48 oz.), and in one case less (viz. 19 oz.).

It appears, therefore, that usually more urine is passed upon farinaceous diet than upon full diet,—often, indeed, considerably more, but there is not much to choose between farinaceous and milk.

Farinaceous Diet combined with Fish or Chicken.

In addition to the cases just mentioned, we have Case 1 (second trial); in this 40 oz. a day were passed on farinaceous diet, against 50 oz. when fish and two eggs were added, and 41 oz. when fish, two eggs, and chop were added. In Case 3 also there was a slight increase

on the addition of chicken, but more on the addition of chicken and fish. In Case 4 the addition of chicken or fish sometimes increased, sometimes diminished, the quantity secreted. In Cases 5 and 8 rather less was passed when fish was added to the farinaceous diet. We learn, therefore, that the addition of fish or chicken or both to the farinaceous diet has very little influence on the amount of urine secreted, and that the influence is uncertain.

Full Diet.—Cases 1, 2, 4, 5, 6, 7.

Case 1 passed 57 oz. a day on full diet, against 75 oz. on farinaceous.

Case 2 passed 40 oz. a day on full diet, against 42 oz. on milk.

Case 4 passed 39 oz. a day on full diet, against 43 oz. previously on farinaceous with fish, and 47 oz. subsequently on farinaceous, and on a second occasion, 21 oz. on full against 26 oz. previously on farinaceous, and 20 oz. subsequently on farinaceous with fish.

Case 5 passed 55 oz. a day on full diet, against 36 oz. previously on farinaceous.

Case 6 passed 52 oz. a day on full diet, against 100 oz. previously on farinaceous, and 88 oz. on milk.

Case 7 passed 42 oz. a day on full diet, against 49 oz. previously on milk.

Result.—In six instances the urine was distinctly less on a full diet than on farinaceous or milk, the decrease being 18, 2, about 6, 5, 48, 7 oz. in each respective case. In two cases there was an increase, but in one of these (Case 2) the urine, which was 21 oz. on a full diet, became 20 oz. on farinaceous and fish, but this trifling difference is probably unimportant, as the patient was dying. In the other case there was an increase of 19 oz. on a full diet, but in this case the patient was improving in all respects. We may conclude, therefore, that as a rule, less urine is passed upon full than upon milk or farinaceous diets.

Full Diet combined with Chop or Eggs.

Case 2. On the addition of a chop and sometimes an egg to full diet, 5 oz. less urine a day were secreted than on milk diet, and 3 oz. less than on full.

Case 7. On the addition of an egg to full diet, 2 oz. less urine were secreted than on full diet only.

When, therefore, chop or eggs were added to full diet, less urine was secreted.

General result.—We may, I think, conclude that usually, in chronic interstitial nephritis, more urine is secreted upon milk and farinaceous diets than upon full diet.

(B) *Two Cases of Chronic Parenchymatous Nephritis.*

Case 9. The secretion was 74 oz. a day on farinaceous diet; it fell to 35 oz. on full.

Case 10. The secretion was 72 oz. a day on farinaceous diet; it fell to 65 oz. on full, rose again to 69 oz. on milk, but fell again to 50 oz. on full.

Result.—The results just enunciated for chronic interstitial nephritis appear to apply to the parenchymatous variety also.

II. The Specific Gravity of the Urine.

The diets are arranged in an ascending scale, that on which the specific gravity of the urine was lowest being placed first.

(A) *Eight Cases of Interstitial Nephritis.*

Case 1. (First trial,) farinaceous, full. (Second trial,) farinaceous and farinaceous with fish and eggs equal, farinaceous with chop, eggs, and fish.

Case 2. Milk, full, and full with chop and egg equal.

Case 3. Milk, farinaceous, farinaceous with chicken or fish.

Case 4. Farinaceous with fish, farinaceous, full, farinaceous with fish, farinaceous with cocoa and full equal, farinaceous with fish, farinaceous with chicken.

Case 5. Full, farinaceous with milk, farinaceous with fish, farinaceous.

Case 6,. Farinaceous, milk, full.

Case 7. Milk, full with egg, full.

Case 8. Farinaceous, farinaceous with fish.

Result.—We may, I think, conclude that the diet has no certain influence on the specific gravity of the urine in chronic interstitial nephritis, but that on the whole it is lower on milk and farinaceous diets than on full diet.

(B) *Two Cases of Chronic Parenchymatous Nephritis.*

Case 9. Farinaceous, full.

Case 10. Farinaceous, milk, full.

Result.—Probably the same is true here also.

III. The Amount of Albumen passed.[1]

(A) *Eight Cases of Interstitial Nephritis.*

Milk Diet.—Cases 2, 3, 6, and 7.

Case 2 passed 8·6 grains a day on milk diet, against 9 grains on full with chop and sometimes egg. (The figure obtained for the three days on full diet is so widely different, that as the days are so few it had better be neglected.)

Case 3 passed 24·3 grains a day on milk diet, against 17·8 grains on farinaceous, 23·8 grains on farinaceous with chicken, and 25 grains on farinaceous with chicken and fish.

Case 6. After the first day in the hospital there was no albumen on milk, farinaceous, or farinaceous with fish diets.

[1] The albumen was always estimated by means of Esbach's tubes. The results are therefore too low ('Clinical Diagnosis,' v. Jaksch, translated by J. Cagney, 2nd edit., p. 263), but my object has not been to ascertain precisely the weight of albumen passed on different diets, but to find out whether altering the diet altered the amount of albumen passed. As Esbach's tubes were used consistently throughout each case they will show this, even although the total amount of albumen registered by them is too low. The exact estimation of albumen is very laborious, and is hardly necessary, as the degree of albuminuria is of very little importance in Bright's disease.

Case 7. No quantitative estimations of albumen were made, but the patient first had milk diet, then full, then full with egg; but the albumen, which was most on milk diet, steadily diminished on the other diets.

Result.—From these cases we learn that milk diet has little or no effect in reducing albuminuria. Sometimes, indeed, more albumen is passed upon milk than upon other diets. When we consider that full diet contains about 400 grains more proteid than either milk or farinaceous, the conclusion seems inevitable that the diet least suitable for compensating for the loss of albumen to the body owing to that in the urine is a milk diet.

Farinaceous Diet.—Cases 1, 3, 4 (two trials), 5, 6, 8.

Case 1 passed 78 grains a day on farinaceous diet, against 23 grains subsequently on full.

Case 3 passed 17·8 grains a day on farinaceous diet, against 24·3 grains on milk, and about 24 grains on farinaceous with chicken or fish.

Case 4 (first trial) passed 39·3 grains a day on farinaceous diet, against 38·3 grains previously on full, 16·8 grains previously on farinaceous with fish, and 37·5 grains on farinaceous with fish or chicken.

Case 4 (second trial) passed 60 grains a day on farinaceous diet, against previously the amounts just mentioned, and subsequently 33 grains on full, and 16·8 grains on farinaceous with fish.

Case 5 passed 162 grains a day on farinaceous diet, against 76 grains on farinaceous with milk, 170 grains on farinaceous with fish, and 66 grains on full.

Case 6 passed no albumen on either farinaceous or full diet.

Case 8 passed 327 grains a day on farinaceous, against 513 grains on farinaceous with fish.

Result.—Contrast with milk: In the one case in which farinaceous can be contrasted with milk diet 6·5 grains of albumen less each day were passed on farinaceous than on milk.

Contrast with full: In the four cases in which farinaceous can be contrasted with full diet, 55, 1, 27, and 96 grains of albumen more each day were passed on farinaceous than on full. The very important result, therefore, follows that albuminuria in chronic interstitial nephritis is much more severe on a farinaceous than on a full diet. As the full diet is much more rich in proteid than farinaceous, the compensation for the albumen lost by the urine is probably very much better on full than on farinaceous diet.

Farinaceous combined with Fish or Chicken.

In addition to the cases just mentioned we have Case 1 (second trial) in which 485 grains of albumen a day were passed on farinaceous diet, against 671 grains on farinaceous with fish and two eggs, and 690 on farinaceous with fish, two eggs, and chop. It will be noticed that in some cases on the addition of fish or chicken the albuminuria increased, in others it diminished; but it increased oftener, and increased more than it decreased.

Full Diet.—Cases 1, 2, 4, 5, 6, 7.

Case 1 passed 23 grains a day on full diet, against 78 grains on farinaceous.

Case 2. For the reason already mentioned the three days on full diet are not available for contrast.

Case 4 passed 38·3 grains a day on full diet, against 16·8 grains previously on farinaceous with fish, and 39·3 grains subsequently on farinaceous; and on a second occasion 33 grains a day on full diet, against 60 grains previously on farinaceous, and 16·8 grains subsequently on farinaceous with fish.

Case 5 passed 66 grains a day on full diet, against 162 grains previously on farinaceous.

Case 6. No albumen on either full or farinaceous diets.

Case 7. The albumen was less on full diet than on milk.

Result.—In only two instances was there any increase of albumen on full diet; both these occurred in Case 4, once soon after admission and once before death: it is

highly probable that these two instances are not of much importance, for in the same case when the conditions were steadier there was a considerable diminution of albumen on full diet. In all the other cases of chronic interstitial nephritis the albumen was less on a full diet than on any other. The importance of this has already been pointed out.

Full Diet combined with Chop or Eggs.

Case 2. The albumen was practically the same on full diet with chop, and sometimes egg, as on milk.

Case 7. The albumen continued to decrease when the egg was added to the full diet.

Result.—These cases appear to show that chop or eggs do not increase the albuminuria in chronic interstitial nephritis.

(B) *Two Cases of Chronic Parenchymatous Nephritis.*

Case 9. In this nearly twice as much albumen was passed upon full diet as upon farinaceous.

Case 10. First occasion, nearly four times as much albumen was passed upon full diet as upon farinaceous. Second occasion, rather less (about $\frac{1}{8}$) was passed on full diet than upon milk.

Result.—It appears that in chronic parenchymatous nephritis the albuminuria is more profuse upon a full diet than upon farinaceous. But full diet contains nearly 400 grains a day more proteid than farinaceous. Now the increased albuminuria on full diet was, in Case 9, 81 grains a day, and in Case 10, 202 grains a day, so that both these patients were probably far better able to compensate for the loss of albumen by the urine upon a full diet than upon farinaceous. This, of course, could not be proved unless the fæces were analysed, but as the full diet did not lead to diarrhœa it is probable that as regards saving of albumen the patients were far better off on full diet than on any other.

IV. The Quantity of Urea passed.

(A) *Eight Cases of Interstitial Nephritis.*

Milk Diet.—Cases 2, 3, 6, and 7.

Case 2 passed 212 grains a day on milk diet, against 176 on full.

Case 3 passed 352 grains a day on milk diet, against 266 on farinaceous, and 289 on farinaceous with chicken.

Case 6 passed 264 grains a day on milk diet, against 397 on farinaceous and 416 on full.

Case 7 passed 363 grains a day on milk diet, against 338 on full, and 344 on full with an egg.

Result.—In two cases much more urea was passed on a milk than upon other diets, but both these cases were going downhill, and as the milk happened to be the first tried, it is quite possible that the diminution in the excretion of urea simply meant that the patient was nearing his end. In one of the other two cases the urea was 133 grains a day less on milk than on farinaceous, and 152 less on milk than on full; in the other the diet made very little difference to the excretion of urea. On the whole it is difficult to say from these cases what is the effect of milk diet on the excretion of urea.

Farinaceous Diet.—Cases 1, 3, 4 (two trials), 5, 6, 8.

Case 1 passed 212 grains a day on farinaceous diet, against 308 on full.

Case 3 passed 266 grains a day on farinaceous diet, against 352 on milk, 289 on farinaceous with chicken, and 283 on farinaceous with chicken and fish.

Case 4 passed 248 grains a day on farinaceous diet, against 223 on full, 275 on farinaceous with fish, and 192 on farinaceous with chicken. On a second occasion 215 grains a day were passed on farinaceous, against 153 on full and 131 on farinaceous with fish.

Case 5 passed 225 grains a day on farinaceous diet,

against 138 on farinaceous with milk, 294 on farinaceous with fish, and 314 on full.

Case 6 passed 397 grains a day on farinaceous diet, against 264 on milk, and 416 on full.

Case 8 passed 209 grains a day on farinaceous diet, against 243 on farinaceous with fish.

Result.—Contrast with milk: In the one case in which this is possible 133 grains a day more urea were passed upon farinaceous than upon milk diet. Contrast with full: In three cases in which this contrast can be made 96, 19, and 89 grains a days less were passed upon farinaceous than on full, and in two cases 25 and 62 grains a day more urea were passed upon farinaceous than upon full. We thus see that in contrasting full with farinaceous diet there is no certain influence upon the output of urea.

Farinaceous combined with Fish or Chicken.

In addition to the cases just mentioned we have Case 1 (second trial), in which 212 grains of urea a day were passed on farinaceous diet, against 376 grains on farinaceous with fish and two eggs, and 235 grains on farinaceous with fish, eggs, and chop. This case shows how uncertain is the effect of adding proteids to farinaceous diet; and Case 4 shows this even more markedly, for sometimes the addition caused an increase and sometimes a diminution of the urea excreted. Cases 3, 5, and 8 passed more urea when proteids were added to farinaceous diet than they did on farinaceous diet only. Although it is not a constant result, the addition of proteids to farinaceous diet usually increases the excretion of urea.

Full Diet.—Cases 1, 2, 4, 5, 6, 7.

Case 1 passed 308 grains a day on full diet, against 212 grains on farinaceous.

Case 2 passed 176 grains a day on full diet, against 212 grains on milk.

Case 4 passed 223 grains a day on full diet, against

248 grains on farinaceous, 275 grains on farinaceous with fish, and 192 grains on farinaceous with chicken. On a second occasion 153 on full, against 215 grains on farinaceous, 154 on farinaceous with cocoa, and 131 on farinaceous with fish.

Case 5 passed 314 grains a day on full diet, against 225 grains on farinaceous, 294 grains on farinaceous with fish, and 138 grains on farinaceous with milk.

Case 6 passed 416 grains a day on full diet, against 397 grains on farinaceous, and 264 grains on milk.

Case 7 passed 338 grains a day on full diet, against 363 grains on milk.

Result.—Contrast with milk: In both cases in which this could be made, actually less urea was passed upon full diet (viz. 36 and 25 grains a day) than upon milk.

Contrast with farinaceous: In three cases more urea (viz. 96, 89, and 19 grains a day) was passed upon full diet than upon farinaceous, but in two cases less urea (viz. 25 and 62 grains a day) was passed upon full diet. The effect, therefore, of diet upon the excretion of urea in chronic interstitial nephritis is most uncertain, for it by no means follows—in fact, the reverse is often true, —that more proteid in the food means more urea excreted.

Full Diet combined with Chop or Eggs.

Case 2. The urea was a few grains less a day when chop and egg were added to the full diet than on full diet only.

Case 7. The urea was a few grains more every day when an egg was added to the full diet than upon a full diet only.

Result.—As far, therefore, as these two cases go, the addition of a chop or egg to the full diet has no noteworthy effect.

(B) *Two Cases of Chronic Parenchymatous Nephritis.*

Case 9. The daily excretion of urea was 382 grains on farinaceous diet, against 302 grains on full.

Case 10. The urea excreted was 421 grains a day on milk diet; 380 grains on one set of days, and 339 grains on another set on full; and 269 grains a day on farinaceous.

Result.—In this variety of nephritis, as in chronic interstitial nephritis, the urea excreted is often less the more proteid the diet contains.

V. The General Condition of the Patient.

The points to which attention was particularly directed in estimating the general condition of the patient were his strength and feeling of well-being, whether he was listless and liked to remain in bed, or whether he wished to get up; his breathlessness and the condition of his circulation.

(A) *Eight Cases of Interstitial Nephritis.*

Case 1 (first admission). For the first nine days on farinaceous diet, for the remaining sixteen on full. He left the hospital having improved considerably in every respect.

Case 1 (second admission). He always felt better and stronger on farinaceous diet with meat and egg added, than on farinaceous.

Case 2. Although this patient ultimately died from cardiac failure associated with valvular disease, he most undoubtedly felt better and stronger on abundant full diet than on milk.

Case 3. There is a note made when the patient had been for some days taking fish or chicken with his farinaceous diet, to say that he certainly felt better on this combination than on milk.

Case 4. The only time that this patient felt better and was able to be up and about was from May 7th to May 16th, for the first four days of which period he was on farinaceous diet, and for the remainder of which he was on full diet.

Case 5. Excepting for three days, when he had a little fish with his farinaceous diet, he had farinaceous only from May 25th to June 20th. From June 15th to June 20th he had severe uræmic symptoms, for which he was treated in the usual way. On June 21st he was put on full diet; from this time he began to gradually improve. He remained on this diet till July 24th, when he was able to leave the hospital.

Case 6. The patient was kept on milk diet for the first three days, farinaceous for the next nine, and full for the last twelve. She steadily improved during the whole of her stay in the hospital.

Case 7. He was on milk diet for the first six days, and on full for the remaining twenty-one. He left the hospital very much better than on admission.

Case 8. This patient was quite certain that she felt very much better on farinaceous with fish than on farinaceous only.

Result.—The testimony of these eight cases is unanimous that the patients feel better and stronger on full diet, or on farinaceous diet with fish or chicken added, than they do on milk or farinaceous diets. Four of the cases died, but the first was from bronchitis due to fog, the second from cardiac failure, both the aortic and mitral valves being diseased, the third had much bronchitis and some pneumonia, the fourth went steadily downhill although several diets were tried, but curiously during the greater part of the only period for which he mended a little he was taking full diet. We see, therefore, that there is no evidence from these cases that a fatal result is brought about by full diet or the addition of meat to farinaceous diet. Nor do these diets induce uræmia or any way weaken the patient. Case 5 is very striking in this respect, for although he had been taking farinaceous diet for nearly four weeks he developed severe uræmia; the very day the symptoms of this abated he was put upon full diet, but they never returned, and he left the hospital greatly relieved. Not only has full diet no tendency to

produce uræmia, but the patients always feel better and stronger on it, or when meat is added to their farinaceous diet. I have often seen this in other cases than those here recorded; some have been instances of interstitial, some of parenchymatous nephritis. Frequently the patients implore and beg for some meat, and my experience is that farinaceous diet is most unpopular in chronic Bright's disease; and, like many others, I have often seen an absolute loathing of milk such as is hardly met with in other maladies.

(B) *Two Cases of Chronic Parenchymatous Nephritis.*

Case 9. This patient did not seem to care what food he took.

Case 10. This patient was quite certain that he felt better on full diet than on milk or farinaceous.

Result.—These cases certainly, as far as they go, bear out the conclusions at which we had arrived concerning chronic interstitial nephritis.

General Conclusions as regards Chronic Bright's Disease.

We see that on the whole an ordinary full diet is the best for this malady, for—(1) It does not increase the liability to uræmia.

(2) The general condition of the patients improves upon it, and they feel stronger and their circulation is better when they are taking it than when they are on milk or farinaceous diet.

(3) A saving of albumen to the body is effected by it, for even if the output of albumen is increased, which it very often is not, more than sufficient extra proteid is taken on the full diet to compensate for any extra loss in the urine, assuming, and there is no evidence to the contrary, that more proteid is absorbed upon full diet than upon milk.

(4) The effect of diet on the excretion of urea is too uncertain to be any guide to us, but there is no evidence that in this respect full diet is harmful.

(5) There is no evidence that full diet contains, or specially leads to the formation of any toxic principles which are harmful in chronic Bright's disease.

(6) This diet prevents the repugnance felt by these patients to farinaceous diet, and their loathing of milk.

The only thing against it is that rather less urine is passed upon full diet than upon milk or farinaceous diet. But this difficulty can be overcome by drinking an extra quantity of water daily, and it must be remembered that in many cases of chronic Bright's disease diuresis is already profuse. It may be well to point out that as the ten cases which form the basis of this paper were all patients who were sufficiently ill to require admission to the hospital, it is quite possible that some of the conclusions might not apply to patients who had Bright's disease in a mild form, or to those accustomed to over-eat themselves.

Clinical History of the Ten Cases of Chronic Bright's Disease, with Analyses of the Urine in each.

Case 1. *Chronic interstitial nephritis; fatal.*—Charles P—, æt. 48, admitted into Guy's Hospital, July 29th, 1891. Clinical clerks, Messrs. G. F. Still and C. S. Pantin.

The patient has often attended at the out-patient department for bronchitis and dyspnœa. He was in the hospital under Dr. F. Taylor for this in November, 1890, and again under Dr. Goodhart in March, 1891, when his symptoms were thought to be due to chronic interstitial nephritis. Since he left the hospital he has had to get up in the night to pass his water, his feet and legs have swollen, and he has had much dyspnœa.

On admission.—There are tophi in his ears. There is much dyspnœa together with physical signs of emphysema. The cardiac dulness extends to one inch outside the nipple. The pulse is not hard. He was discharged on August 30th, having improved considerably in all respects.

Second admission.—He was readmitted on October 6th. At first, after he left the hospital last time, he seemed very

well, and was able to continue his work as a travelling photographer. He got wet, and soon after that he noticed that his urine was very scanty, that his legs and abdomen began to swell considerably, and that he had much dyspnœa.

On admission.—There was much œdema, and great dyspnœa with râles and rhonchi over both lungs. Pulse 64, not hard. The diagnosis made was subacute Bright's disease supervening upon chronic interstitial nephritis. His chief trouble was the bronchitis and pleuritic effusion; the latter was relieved by aspiration. He always felt better and stronger when he was on full diet than farinaceous. It appeared that the dense fog which prevailed in December increased his bronchitis to a fatal extent.

Autopsy.—There was found chronic interstitial nephritis and a greatly hypertrophied heart. The lungs were emphysematous.

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
July							
29	20	—	—	—	—	—	Farinaceous.
30	29	—	6·0	76·1	—	—	
31	67	—	3·0	87·9	—	—	
Aug.							
1	92	1012	3·0	120·7	1·0	402	
3	114	1008	1·5	74·7	0·3	149	
4	104	1012	1·5	68·2	0·4	182	
5	104	1010	1·5	68·2	0·4	182	
6	67	1008	—	—	0·5	146	
7	77	1010	1·5	50·5	—	—	
8	48	1010	2·0	42·0	0·9	188	Full.
10	50	1012	1·4	30·6	0·7	153	
11	56	1014	1·8	44·1	1·0	245	
12	56	1014	1·2	29·4	0·8	196	
13	51	1015	—	—	1·2	267	
14	46	—	1·0	20·1	1·5	301	
15	46	1014	1·0	20·1	1·4	281	
17	46	1014	1·0	20·1	1·6	322	
18	57	1014	0·8	19·9	1·4	349	
19	70	1013	0·8	24·5	1·3	398	
20	67	1012	0·8	23·4	1·3	381	

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
Aug.							
21	72	1015	0·8	25·2	1·5	472	Full.
23	70	1014	0·5	15·3	1·4	428	
24	69	1012	0·5	15·0	—	—	
26	57	1013	0·5	12·4	1·3	324	
27	57	1014	0·5	12·4	1·3	324	
Oct.							
6	—	1020	50·0	—	1·6	—	Farinaceous.
7	—	1015	30·0	—	1·8	—	
8	28	1015	30·0	367·3	1·6	196	
9	48	1012	20·0	420·0	1·5	314	
10	50	1012	20·0	437·5	1·2	262	
12	64	1010	30·0	839·6	0·8	224	
13	24	1020	—	—	1·5	157	
14	16	1015	—	—	1·2	84	
15	54	1015	22·5	511·5	—	—	
16	36	1015	21·2	333·9	1·6	252	
17	44	1019	22·5	433·0	1·6	308	Farinaceous with fish
19	54	1016	30·0	708·4	2·0	472	and two eggs.
20	48	1017	37·5	787·4	2·2	462	
21	52	1016	33·5	762·1	2·0	455	
22	44	1016	25·0	699·5	2·1	587	
23	36	1018	—	—	2·1	330	
26	52	1018	27·5	625·6	1·8	409	
27	48	1008	22·5	472·4	0·7	146	
28	48	1016	40·0	840·0	1·6	336	
29	64	1014	30·0	840·0	1·6	448	
30	48	1014	—	—	1·3	273	
31	50	1014	25·0	546·5	1·3	284	
Nov.							
2	72	1012	17·5	551·2	1·1	346	Farinaceous with
3	48	1013	—	—	1·5	314	fish, chop, and two
4	48	1011	20·0	420·0	1·7	357	eggs.
5	40	1018	—	—	1·7	297	
6	52	1017	—	—	1·7	386	
7	28	1020	37·5	459·3	1·8	220	
9	44	1019	50·0	962·2	1·8	346	
10	—	1020	67·5	—	1·1	—	
11	—	1016	37·5	—	2·0	—	
13	40	1016	—	—	1·8	314	
14	40	—	25·0	437·2	—	—	
16	34	1018	32·5	483·4	2·1	312	
17	40	1018	32·5	568·7	1·8	314	
18	44	1018	50·0	962·2	1·1	211	
19	44	1020	55·0	1058·6	1·8	346	
20	44	1018	40·0	770·0	1·1	211	
24	76	1014	—	—	1·3	432	
25	72	—	70·0	2205·0	—	—	
26	40	1015	45·0	787·2	1·3	227	
30	36	1014	15·0	236·1	—	—	

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
Dec.							
1	24	1018	55·0	557·4	1·8	188	Farinaceous, with fish, chop, and two eggs.
2	8	1020	52·5	183·7	1·2	42	
3	24	1014	52·5	551·2	1·6	168	
4	68	1016	40·0	1190·0	1·7	506	
5	16	1018	47·5	332·4	1·8	127	
7	40	1014	—	—	1·8	314	
8	44	1018	37·5	721·8	1·7	327	
9	36	1018	47·5	748·1	1·8	283	
10	36	1018	—	—	2·3	262	
11	36	1018	52·5	826·8	2·0	315	
14	68	1012	—	—	1·4	416	
15	28	1006	40·0	490·0	2·0	245	
16	26	1018	47·5	540·3	2·2	250	
17	28	1018	37·5	459·3	1·9	232	
18	32	1012	—	—	1·8	251	

Summary of Quantity of Urine passed.

		Total quantity. Ounces.		Daily average. Ounces.
9 days of	farinaceous diet . . .	674	...	75
16 „	full diet	918	...	57
8 „	farinaceous diet . . .	320	...	40
12 „	„ fish, and 2 eggs .	608	...	50
33 „	„ fish, 2 eggs, and chop	1356	...	41

Summary of Specific Gravity.

		Daily average.
6 days of	farinaceous diet	1010
15 „	full diet	1013
10 „	farinaceous diet	1015
12 „	„ fish, and 2 eggs . . .	1015
33 „	„ fish, 2 eggs, and chop . .	1016

Summary of Quantity of Albumen passed.

		Total quantity. Grains.		Daily average. Grains.
7 days of	farinaceous diet . . .	546	...	78
15 „	full diet	354	...	23
6 „	farinaceous diet . . .	2910	...	485
10 „	„ fish, and 2 eggs .	6715	...	671
24 „	„ fish, 2 eggs, and chop	16522	...	690

SUMMARY OF QUANTITY OF UREA PASSED.

		Total quantity. Grains.		Daily average. Grains.
5 days of	farinaceous diet . . .	1061	...	212
15 „	full diet	4629	...	308
7 „	farinaceous diet . . .	1489	...	212
12 „	„ fish, and 2 eggs .	4511	...	376
30 „	„ fish, 2 eggs, and chop	8559	...	285

N.B.—As the patient was out of the hospital for five weeks between the full diet and the second farinaceous, they should not be contrasted.

CASE 2.—*Chronic interstitial nephritis; fatal.*—Thomas M—, æt. 44, admitted into Guy's Hospital October 15th, 1890.

He has had gout for the last four or five years. He was in the Greenwich Hospital three months ago with swelling of the face, legs, and genitals. He has at times had fits. He has passed blood in his urine.

On admission.—There are tophi in his ears, and his hands, wrists, and knees are deformed by gout. His face is œdematous. The position of the apex of the heart is normal; the first sound is rough, the second is accentuated; the pulse is hard, 94. The abdomen contains a little fluid; the eyes are normal.

October 20th.—Has had vomiting and diarrhœa the last few days; a systolic murmur has been audible at the apex. Has been treated with compound jalap powder and subcutaneous injections of nitrate of pilocarpine.

Up to November 10th he had milk diet, jalap, and pilocarpine as required, and perchloride of iron thrice a day; from this date he was put upon colchicum and full diet. He was able to get up on November 14th, and began to steadily improve in all respects, so that by December 6th there was marked improvement, and he felt much stronger. About this time symptoms of cardiac failure again began to show themselves, and the patient gradually sank, and died on December 24th.

Post-mortem.—The aortic valves were thickened and everted, the mitral valve was thick. The heart was

hypertrophied. The kidneys only weighed 5½ oz. They showed typical interstitial nephritis with much tubal change.

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
Oct.							
17	48	1010	1·0	21·0	1·0	230	Milk.
20	48	1008	0·5	10·4	1·0	230	
21	44	1010	0·5	9·6	1·6	337	
22	16	1010	0·5	3·5	1·2	92	
23	38	1010	0·5	8·3	1·0	182	
24	40	1010	0·25	4·3	1·0	192	
25	36	1010	0·5	7·8	1·4	241	
26	36	1010	0·5	7·8	—	—	
27	38	1010	0·5	8·3	1·1	201	
28	36	1010	0·4	6·3	1·0	172	
29	24	1010	0·5	5·2	1·4	161	
30	26	1010	0·5	5·6	1·2	149	
31	36	1010	0·5	7·8	1·2	207	
Nov.							
1	42	1010	0·5	9·1	1·35	272	
2	40	1010	—	—	—	—	
3	32	1010	0·5	7·0	1·1	168	
4	32	1010	0·5	7·0	1·0	153	
5	36	1010	0·5	7·8	1·0	172	
6	40	1010	0·5	8·7	1·0	192	
7	46	1010	0·5	10·0	0·9	198	
8	68	1010	0·5	14·9	0·9	293	
9	98	1010	0·5	26·4	—	—	
10	70	1010	0·5	15·3	1·15	386	
11	52	1008	0·3	6·8	0·9	224	
12	60	1010	0·3	7·8	0·9	259	Full diet, with one chop, and sometimes eggs.
13	42	1010	0·5	9·1	1·1	221	
14	50	1010	0·5	10·9	0·9	216	
15	36	1010	0·5	7·8	1·4	241	
16	52	—	—	—	—	—	
17	20	1010	0·5	4·3	0·9	86	
18	50	1010	0·5	10·9	1·2	288	
19	48	1010	0·5	10·4	0·8	184	
20	32	1010	0·5	7·0	0·9	138	
21	40	1010	0·5	8·7	0·9	172	
22	28	1010	0·5	5·1	0·8	107	
23	34	1010	0·5	7·4	0·9	146	
24	38	1010	0·5	8·3	0·9	164	
25	40	1010	0·6	10·5	0·8	153	
26	38	1010	0·6	8·3	0·9	164	
27	36	1010	0·5	7·8	0·8	138	
28	50	1010	0·5	10·9	0·9	216	
29	36	1010	0·5	7·8	1·4	241	
30	36	1010	0·5	7·8	0·8	138	

Date.	No. of ozs. in 24 hrs	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
Dec.							
1	20	1010	0·5	4·3	0·7	67	Full diet, with one
2	38	1010	0·75	12·4	0·9	164	chop, and sometimes
3	36	1010	0·5	7·8	0·9	155	eggs.
4	36	1010	1·0	15·7	0·8	138	
5	38	1010	1·0	16·6	1·0	182	
6	24	1010	1·0	10·5	0·9	103	
7	32	1010	—	—	—	—	
8	36	1010	1·0	15·7	0·9	155	
9	40	1010	1·0	8·6	1·0	192	
10	38	1010	1·0	16·6	0·8	145	
11	30	1010	1·0	13·1	0·9	129	
12	38	1010	1·0	16·6	0·9	164	Chop discontinued,
13	54	1010	1·0	23·6	0·9	227	otherwise the same.
14	36	—	—	—	—	—	
15	32	1009	1·5	21·0	0·9	138	
16	34	1009	0·5	7·4	0·9	161	Full diet, with chop.
17	42	1010	0·4	7·3	0·9	181	
18	44	1005	—	—	0·9	190	
19	38	1008	0·5	8·3	0·9	164	
20	32	1010	—	—	0·9	138	
21	34	1010	0·4	5·9	0·9	146	
22	32	1009	0·5	7·0	0·9	138	
23	34	1010	—	—	1·0	163	

Summary of Quantity of Urine passed.

	Total quantity. Ounces.	Daily average. Ounces.
24 days of milk diet	1022 ...	42
38 „ full diet, chop, and sometimes egg .	1424 ...	37
4 „ full diet	160 ...	40

Summary of Specific Gravity.

	Daily average.
24 days of milk diet	1009·8
38 „ full diet, chop, and sometimes egg . . .	1009·7
3 „ full diet	1009·6

Summary of Quantity of Albumen passed.

	Total quantity. Grains.	Daily average. Grains.
23 days of milk diet	198 ...	8·6
33 „ full diet, chop, and sometimes egg .	297 ...	9·0
3 „ full diet	61 ...	20·4

SUMMARY OF QUANTITY OF UREA PASSED.

	Total quantity. Grains.		Daily average. Grains.
21 days of milk diet	4452	...	212
36 „ full diet, chop, and sometimes egg .	6043	...	168
3 „ full diet	529	...	176

CASE 3. *Chronic interstitial nephritis; fatal.*—Samuel G—, æt. 38, admitted into Guy's Hospital April 8th, 1892. He is a painter. Clinical clerk, Mr. F. G. M. Phelps.

About last Christmas he began to suffer from cough and shortness of breath. This has got worse, and at the same time his feet and genitals have become œdematous. He has to get up at night to pass his water.

On admission.—Is very anæmic; there is much œdema and ascites. The apex-beat is in the nipple line, and the pulse is very hard and tension high. There is impaired resonance at the bases of both lungs, and there are a few moist râles in the chest. Albuminuric retinitis is present.

During his life the bronchitis troubled him much. Considerable fluid collected in his chest, which was aspirated. Southey's tubes were put in the legs. He suffered much from dyspnœa, and towards the end he was drowsy.

Vapour-baths, pilocarpine, and jalap powder were all employed. Although in spite of treatment these organic symptoms progressed, there is a note on April 28th to say "Patient has certainly felt better since the milk diet was changed." He died May 13th.

Post-mortem.—There was well-marked chronic interstitial nephritis and some pneumonia in the lungs.

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
April							
11	28	1010	1·0	12·2	—	—	Milk.
12	60	1010	0·75	20·0	—	367	
13	64	1010	1·5	42·0	—	364	
14	58	1008	1·0	25·0	—	336	
15	60	1010	1·0	26·0	—	—	
16	60	1008	0·75	21·0	—	341	
18	48	1008	1·2	25·0	—	—	Farinaceous.
19	40	1010	1·4	24·5	—	336	
20	—	—	—	—	—	245	
21	20	1012	1·25	10·0	—	—	
22	32	1010	1·25	17·5	—	252	
23	28	1012	1·0	12·2	—	232	
25	38	1012	1·0	16·6	—	299	Farinaceous with fish and arrowroot.
26	36	1012	1·25	19·6	—	283	Farinaceous with chicken.
27	—	1012	1·5	—	—	—	
28	28	1013	2·0	24·5	—	245	
29	36	1012	1·75	27·5	—	299	
30	40	1012	—	—	—	332	
May							
2	48	1012	1·5	31·5	—	399	Farinaceous with fish and chicken.
3	48	1012	1·5	31·5	—	378	
4	36	1012	1·75	27·5	—	315	
5	34	1010	2·0	29·7	—	282	
6	55	1012	2·0	48·1	—	481	
7	36	1012	2·0	31·5	—	—	
9	20	1013	1·75	15·3	—	183	
10	24	1013	1·75	18·3	—	220	
11	34	1013	1·5	22·3	—	297	
12	12	1012	1·5	7·8	—	105	
13	20	1012	1·25	10·9	—	175	

Summary of Quantity of Urine passed.

	Total quantity. Ounces.		Daily average. Ounces.
6 days of milk diet	330	...	55
5 „ farinaceous diet . . .	168	...	33
4 „ „ and chicken .	140	...	35
11 „ „ chicken, and fish .	367	...	33

SUMMARY OF SPECIFIC GRAVITY.

			Daily average.
6 days of	milk diet		1009·3
5 „	farinaceous diet		1010·4
5 „	„	and chicken	1012·2
11 „	„	chicken, and fish	1012·1

SUMMARY OF QUANTITY OF ALBUMEN PASSED.

			Total quantity. Grains.		Daily average. Grains.
6 days of	milk diet		146·2	...	24·3
5 „	farinaceous diet		89·2	...	17·8
8 „	„	and chicken	71·6	...	23·8
11 „	„	chicken, and fish	274·4	...	25·0

SUMMARY OF QUANTITY OF UREA PASSED.

			Total quantity. Grains.		Daily average. Grains.
4 days of	milk diet		1408	...	352
4 „	farinaceous diet		1065	...	266
4 „	„	and chicken	1159	...	289
10 „	„	chicken, and fish	2835	...	283

CASE 4. *Chronic interstitial nephritis; fatal.*—Thomas H—, æt. 47, admitted into Guy's Hospital 15th March, 1892. He has often had gout. Clinical clerk, Mr. G. A. Skinner.

On admission.—There is œdema of the eyelids. The cardiac dulness is increased, and the apex is outside the nipple. The pulse is high tension. There is a systolic bruit audible at the apex. There are retinal hæmorrhages and albuminuric retinitis. The patient never improved much; he suffered considerably from headache and dyspnœa. The retinitis progressed rapidly. The only time he improved was from May 7th to May 16th, when he felt a little better, and was able to be up; but as the œdema reappeared, he had to go back to bed. On May 21st; 32 oz. of clear fluid were withdrawn from each chest, and this was done again a week later. On May 28th he became maniacal and sank, and died on June 3rd. Various diuretics and diaphoretics were tried, but none of them did any good.

Post-mortem.—There was found chronic interstitial nephritis, an hypertrophied heart, and gummata in the liver.

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
March							
17	44	1010	—	17	—	—	Fish.
18	44	1014	—	17	—	—	
19	44	1015	—	17	—	—	
20	44	1015	—	17	—	—	
21	40	1015	—	16	—	—	
22	40	1012	—	40	1·5	262	Full.
23	30	1014	—	27	1·2	157	
24	48	1012	—	48	1·2	252	
25	48	1012	—	96	1·2	252	Farinaceous.
26	48	1014	—	48	1·2	252	
27	32	1012	—	32	1·2	168	
28	48	1012	—	48	1·3	273	
29	50	—	—	50	1·2	262	
30	56	1010	—	56	1·2	294	
31	60	1010	—	60	1·2	315	
April							
1	48	1011	—	31	1·4	294	
2	58	1010	—	32	1·0	253	
3	72	—	—	36	—	—	
4	50	1010	—	—	0·9	196	
5	28	1012	—	12	1·7	208	
6	44	1010	—	33	1·2	231	
7	25	—	—	12	—	—	
8	40	1012	—	20	1·3	227	
9	48	1010	—	24	1·2	252	
11	48	1010	—	36	1·3	273	Farinaceous and fish.
12	56	1010	—	36	1·25	306	
13	56	—	—	36	1·0	245	
14	50	1010	—	32	1·2	262	
15	48	1011	—	46	1·3	273	
16	48	1008	—	31	1·5	314	
18	48	1009	—	46	1·2	252	
19	36	1014	—	40	1·4	220	Farinaceous and chicken.
20	32	1014	—	41	1·5	219	
21	20	1014	—	32	1·8	137	
22	24	1012	—	42	1·8	188	Farinaceous.
23	24	1010	—	—	1·9	199	
25	28	1014	—	67	1·8	220	
26	28	1012	—	70	1·9	232	
27	28	1014	—	57	—	—	
28	32	1012	—	64	1·7	238	
29	36	1012	—	86	—	—	Cocoa.
30	36	1012	—	65	1·4	220	

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
May							
2	41	1010	—	90	1·6	287	Cocoa.
3	12	1012	—	31	1·5	78	
4	12	1016	—	—	1·5	78	
5	20	1014	—	40	1·3	113	
6	20	1015	—	48	1·6	140	
7	32	1015	—	—	—	—	
8	24	—	—	—	—	—	
9	20	1010	—	—	1·7	148	
10	24	1010	—	31	1·6	168	
11	28	1012	—	33	1·5	183	Full.
12	28	1012	—	36	1·9	232	
13	20	1012	—	45	1·6	140	
14	8	1012	—	18	1·7	59	
15	24	—	—	—	—	—	
16	16	1012	—	12	1·5	104	Farinaceous and fish.
17	20	—	—	—	—	—	
18	20	1012	—	19	1·7	148	
19	16	1014	—	14	1·5	104	
20	16	1012	—	16	1·6	112	
21	24	1014	—	18	1·5	157	
22	28	—	—	—	—	—	
23	20	1012	—	19	1·5	131	
24	20	1012	—	17	1·6	140	
25	24	1012	—	21	1·6	168	
27	16	1012	—	16	1·7	119	

Summary of Quantity of Urine passed.

		Total quantity. Ounces.		Daily average. Ounces.
5 days of	farinaceous and fish diet	216	...	43
3 „	full diet	118	...	39
16 „	farinaceous diet	755	...	47
7 „	„ and fish	354	...	50
3 „	„ and chicken	88	...	29
6 „	„	164	...	27
11 „	„ and cocoa	287	...	26
5 „	full diet	108	...	21
11 „	farinaceous diet and fish	220	...	20

SUMMARY OF SPECIFIC GRAVITY.

			Daily average.
5	days	of farinaceous diet and fish	1013·8
3	„	full diet	1012·6
13	„	farinaceous diet	1011·1
6	„	„ and fish	1009·6
3	„	„ and chicken	1014·0
6	„	„	1012·3
10	„	„ and cocoa	1012·6
4	„	full diet	1012·0
9	„	farinaceous diet and fish	1012·4

SUMMARY OF QUANTITY OF ALBUMEN PASSED.

			Total quantity. Grains.		Daily average. Grains.
5	days	of farinaceous diet and fish	84	...	16·8
3	„	full diet	115	...	38·3
15	„	farinaceous diet	590	...	39·3
7	„	„ and fish	263	...	37·5
3	„	„ and chicken	113	...	37·6
5	„	„	300	...	60·0
7	„	„ and cocoa	391	...	55·8
4	„	full diet	132	...	33·0
9	„	farinaceous diet and fish	152	...	16·8

SUMMARY OF QUANTITY OF UREA PASSED.

			Total quantity. Grains.		Daily average. Grains.
3	days	of full diet	671	...	223
14	„	farinaceous diet	3477	...	248
7	„	„ and fish	1925	...	275
3	„	„ and chicken	576	...	192
5	„	„	1077	...	215
8	„	„ and cocoa	1232	...	154
4	„	full diet	614	...	153
9	„	farinaceous diet and fish	1183	...	131

CASE 5. *Chronic interstitial nephritis.*—George R—, æt. 63, admitted into Guy's Hospital May 22nd, 1892. Clinical clerk, Mr. A. L. Roper. The patient has had gout for thirty-three years. Lately he has noticed that his eyelids have been swollen.

On admission.—He has tophi in his ears. His chest is emphysematous. The pulse is hard; the apex-beat cannot be felt.

From June 15th to June 20th, he had severe uræmia, as shown by convulsions, coma, and vomiting. He was treated with vapour-baths, elaterium, and pilocarpine. He gradually got better, and was able to get up on July 5th. Except that for a few days about July 9th he had headache he did well, and was able to leave the hospital on July 24th better in all respects than when he came in.

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
May							
25	20	1010	1·0	8·7	1·5	130	Farinaceous with milk.
26	16	1012	1·0	7·0	1·4	98	
27	20	1012	7·0	61·0	1·6	140	
28	20	1012	7·0	61·0	1·6	140	
30	16	1016	20·0	140·0	1·6	112	
31	28	1018	15·0	178·0	1·8	212	
June							
1	36	1016	12·0	189·0	2·0	317	Farinaceous and fish.
2	32	1020	15·0	208·0	2·0	279	
3	20	1020	13·0	113·0	2·2	192	
10	28	1016	8·0	98·0	1·4	171	Farinaceous.
11	48	1016	12·0	251·0	1·6	336	
12	24	1015	10·0	104·0	1·3	135	
13	32	1012	12·0	168·0	1·2	167	
14	44	1016	12·0	231·0	1·1	211	
15	40	1014	13·5	236·0	1·2	210	
16	—	1016	18·0	—	1·1	—	
17	20	1020	12·0	104·0	1·4	122	
18	50	1020	6·0	130·0	1·8	393	
20	36	1018	9·0	140·0	1·8	285	
21	54	1018	7·5	172·0	2·1	496	Full.
22	30	1016	6·0	78·0	1·8	236	
23	50	1012	6·0	131·0	1·8	393	
24	50	1012	10·5	225·0	1·8	393	
25	48	1012	9·0	187·0	—	—	
27	60	1010	3·0	78·0	1·7	446	
28	50	1012	4·5	98·0	1·3	284	
29	54	1014	3·0	70·0	1·6	378	
30	54	1013	—	—	1·6	378	
July							
1	64	1014	1·5	41·0	1·2	336	
2	36	1012	1·5	23·0	1·4	214	
4	64	1014	1·5	41·0	1·3	373	
5	40	1014	1·5	26·0	1·4	245	
6	56	1014	1·5	36·0	1·3	321	
7	58	1010	2·5	61·0	1·2	304	
8	52	1010	2·5	64·0	1·1	249	
9	80	1008	2·0	70·0	0·8	216	

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
July							
11	64	1010	2·0	56·0	1·1	307	Full.
12	64	1012	2·0	56·0	1·0	279	
13	64	1008	2·0	56·0	1·2	336	
14	68	1010	1·5	44·0	1·1	328	
15	64	1010	1·5	41·0	1·1	307	
16	64	1010	1·0	28·0	1·1	307	
18	45	1010	1·0	17·0	1·2	236	
19	60	1012	1·0	26·0	1·1	288	
20	60	1010	1·0	26·0	1·2	315	
21	60	1010	1·0	26·0	1·2	315	
22	48	1008	0·5	10·0	1·0	209	

Summary of Quantity of Urine passed.

		Total quantity. Ounces.	Daily average. Ounces.
6 days	of farinaceous diet and milk	120	20
3 „	„ and fish	88	29
9 „	„	322	36
28 „	full diet	1561	55

Summary of Specific Gravity.

		Daily average.
6 days	of farinaceous diet and milk	1013·3
3 „	„ and fish	1018·6
10 „	„	1016·3
28 „	full diet	1011·6

Summary of Quantity of Albumen passed.

		Total quantity. Grains.	Daily average. Grains.
6 days	of farinaceous diet and milk	455·7	76
3 „	„ and fish	510	170
9 „	„	1462	162
27 „	full diet	1787	66

Summary of Quantity of Urea passed.

		Total quantity. Grains.	Daily average. Grains.
6 days	of farinaceous diet and milk	832	138
3 „	„ and fish	788	294
9 „	„	2030	225
27 „	full diet	8489	314

Case 6. *Chronic interstitial nephritis.*—Matilda C—, æt. 71, admitted into Guy's Hospital 12th May, 1891. Clinical clerk, Mr. F. Wallace Wilson.

She says she caught cold last winter. A few weeks ago the feet began to swell, and the eyelids to be puffy. Soon after the abdomen began to distend, and she suffered from dyspnœa.

On admission.—There is subconjunctival œdema and puffiness beneath the eyes. The legs are œdematous, and there is a well-marked lumbar cushion. The præcordial dulness is increased, and a distinct pericardial rub is audible. There is also a systolic endocardial murmur, best heard at the apex. The pulse is rapid, weak, and irregular. There is impaired resonance with râles at the bases of both lungs. She was ordered small doses of tincture of digitalis and acetate of ammonium, together with a mustard leaf over the pericardium. The pericardial rub slowly disappeared; the patient improved in all respects, and left the hospital on June 7th, 1891.

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
May							
14	24	1022	Trace	—	—	—	Milk.
15	98	1010	None	—	0·8	343	
16	142	1008	,,	—	0·3	186	
17	152	1009	,,	—	0·45	297	Farinaceous.
18	156	1010	,,	—	0·5	340	
19	132	—	,,	—	—	—	
20	76	1010	,,	—	1·0	338	
21	108	1010	,,	—	0·9	424	
22	96	—	,,	—	0·9	393	
23	72	1012	,,	—	1·4	440	
24	52	—	,,	—	—	—	
25	60	1016	,,	—	2·1	550	
26	80	1010	,,	—	1 3	454	Full.
27	68	1012	,,	—	1·5	446	
28	60	1014	,,	—	2·1	550	
29	48	1016	,,	—	1·9	398	
30	70	1010	,,	—	1·5	459	
31	48	—	,,	—	—	—	
June							
1	40	1018	,,	—	2·6	454	
2	28	1020	,,	—	2·3	281	
3	32	1020	,,	—	2·6	363	
4	48	1014	,,	—	1·9	398	
5	56	1016	,,	—	2·1	503	
6	52	1018	,,	—	1·2	273	

SUMMARY OF QUANTITY OF URINE PASSED.

		Total quantity. Ounces.	Daily average. Ounces.
3 days of	milk diet	264 ...	88
9 „	farinaceous diet . . .	904 ...	100
12 „	full	630 ...	52

SUMMARY OF SPECIFIC GRAVITY.

		Daily average.
3 days of	milk diet	1013
6 „	farinaceous diet	1011
11 „	full diet	1015·2

ALBUMEN.

After the first day there was none on any diet.

SUMMARY OF QUANTITY OF UREA PASSED.

		Total quantity. Grains.	Daily average Grains.
2 days of	milk diet	529 ...	264
7 „	farinaceous diet . . .	2782 ...	397
11 „	full	4579 ...	416

CASE 7. *Chronic interstitial nephritis.*—Thomas R—, æt. 67, admitted into Guy's Hospital 9th May, 1891.

Nine months ago his feet and ankles began to swell, he also had pain in the head, shortness of breath, and cough, and he suffered from indigestion, constipation, and epistaxis.

On admission.—There was much œdema of the feet. The apex of the heart could not be felt, but there was a faint systolic murmur audible in its usual position. The pulse was very hard and 66 to the minute. The breathing was harsh. He was ordered acetate of ammonium. He left the hospital on June 9th, and said he felt better than when he came in.

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
May							
10	—	1008	—	—	—	—	Milk.
11	32	—	—	—	—	—	
12	64	1012	—	—	—	—	
13	48	—	—	—	1·3	273	
14	52	1012	—	—	1·8	409	
15	52	1010	—	—	1·8	409	
16	58	1010	—	—	1·7	431	Full.
17	46	—	—	—	—	—	
18	28	1020	—	—	1·5	183	
19	34	—	—	—	—	—	
20	60	1014	—	—	2·3	603	
21	40	1020	—	—	1·9	332	
22	40	1016	—	—	2·0	350	
23	20	1018	—	—	1·5	131	
25	52	1016	—	—	1·5	341	
26	34	1016	—	—	1·7	252	Full with egg for
27	36	1012	—	—	2·0	315	breakfast.
28	36	1020	—	—	2·6	409	
29	40	1016	—	—	2·4	420	
30	44	1014	—	—	1·8	346	
31	44	—	—	—	—	—	
June							
1	40	1018	—	—	1·9	332	
2	32	1016	—	—	1·9	265	
3	50	1014	—	—	1·8	393	
4	36	1020	—	—	2·0	315	
5	50	1014	—	—	2·0	437	
6	44	1012	—	—	1·6	308	

Blood and albumen present during the whole time, but they diminished towards the end.

Summary of Quantity of Urine passed.

	Total quantity. Ounces.	Daily average. Ounces.
5 days of milk diet	248 ...	49
9 „ full diet	378 ...	42
12 „ „ and egg . . .	486 ...	40

Summary of Specific Gravity.

	Daily average.
4 days of milk diet	1010·5
7 „ full diet	1016·3
11 „ „ and egg	1015·6

SUMMARY OF QUANTITY OF UREA PASSED.

	Total quantity. Grains.	Daily average. Grains.
3 days of milk diet	1091 ...	363
7 „ full diet	2371 ...	338
11 „ „ and egg . . .	3792 ...	344

CASE 8. *Chronic interstitial nephritis.*—Matilda H—, æt. 46, admitted into Guy's Hospital 11th March, 1891. Clinical clerk, Mr. F. H. Evans.

In this case there was no doubt about the diagnosis because she had well-marked albuminuric retinitis. The heart was hypertrophied. She did not stay in the hospital long, but went home at her own wish. She said she was quite sure that she felt much better on fish than on farinaceous diet.

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
March							
11	—	1012	2·7	—	—	—	Farinaceous.
12	—	1012	2·5	—	—	—	
13	48	1012	2·5	525	1·3	273	
14	24	1013	2·3	241	1·3	136	
16	24	1013	2·5	262	1·8	189	
17	46	1015	2·2	441	1·65	332	
18	48	1011	1·5	314	1·1	230	
19	28	1013	2·2	269	1·4	171	
20	24	1013	1·7	178	1·15	120	
21	44	1014	2·0	385	1·15	221	
23	20	1021	5·0	437	1·6	140	Fish.
24	36	1017	2·2	346	1·9	299	
25	36	1018	2·5	393	1·7	267	
26	16	1020	2·5	175	1·5	105	
28	46	1014	2·6	523	1·1	221	
30	32	1016	6·0	840	1·4	196	
April							
1	42	1014	3·0	554	2·1	388	
2	38	1012	5·C	836	2·0	334	

SUMMARY OF QUANTITY OF URINE PASSED.

	Total quantity. Ounces.	Daily average. Ounces.
8 days of farinaceous diet . . .	286 ...	36
8 „ „ and fish . .	266 ...	33

SUMMARY OF SPECIFIC GRAVITY.

	Daily average.
10 days of farinaceous diet	1012·8
8 „ „ and fish	1016·4

SUMMARY OF QUANTITY OF ALBUMEN PASSED.

	Total quantity. Grains.	Daily average. Grains.
8 days of farinaceous diet . . .	2615 ...	327
8 „ „ and fish . .	4104 ...	513

SUMMARY OF QUANTITY OF UREA PASSED.

	Total quantity. Grains.	Daily average. Grains.
8 days of farinaceous diet . . .	1672 ...	209
8 „ „ and fish . .	1950 ...	243

CASE 9. *Chronic parenchymatous nephritis.*—W. R—, æt. 35, admitted into Guy's Hospital October 23rd, 1891. Clinical clerk, Mr. R. H. Luce.

He has had an abscess under the lower jaw, which has been discharging for the last five years. His legs began to swell about a month ago.

On admission.—There is much œdema of the feet and some of the eyelids. The cardiac dulness extends half an inch outside the nipple. Except for these signs and the albuminuria, nothing else abnormal could be found. Neither the liver nor spleen could be felt. It was possible that the kidneys were to some extent lardaceous. The patient left the hospital on November 30th in the same condition as he was when he came in.

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
Oct.							
24	78	1018	—	—	—	—	Farinaceous.
26	78	1011	3·0	102·3	0·95	326	
27	72	1015	2·75	86·6	1·95	617	
28	72	1010	2·5	78·7	0·9	285	
28	72	1010	2·75	86·6	0·95	301	
30	54	1015	5·0	114·1	1·4	332	Full.
31	32	1017	3·0	42·0	1·8	253	
Nov.							
2	48	1014	5·0	105·0	1·8	380	
3	40	1014	5·5	96·2	1·6	281	
4	40	1018	7·0	122·5	1·7	299	
5	24	1022	9·0	94·5	2·3	242	
6	30	1027	12·0	157·5	2·3	303	
7	20	1028	12·0	105·0	2·6	228	
9	24	1025	14·0	147·0	2·0	211	
10	32	1024	14·0	196·0	2·5	320	
11	32	1020	18·0	252·0	2·1	295	
12	30	1025	16·0	210·0	2·7	356	
13	16	1026	28·0	196·0	3·0	217	
14	26	1025	13·5	153·5	3·0	343	
16	24	1022	17·0	178·5	2·9	306	
17	48	1020	13·0	273·0	2·1	443	
18	32	1020	12·0	168·0	1·9	267	
19	40	1022	13·0	227·5	2·2	387	
20	44	1019	8·5	163·6	1·65	319	
21	32	1020	5·0	70·0	2·05	288	
23	30	1022	13·0	170·6	2·2	290	
24	44	1024	12·0	231·0	2·1	369	
25	30	1024	14·0	183·7	2·2	290	
26	24	1022	14·0	147·0	2·4	253	
27	44	1016	15·0	288·7	1·45	280	
28	48	1018	8·0	168·0	1·4	295	
30	60	1012	12·0	315·0	1·2	316	

Summary of Quantity of Urine passed.

	Total quantity. Ounces.	Daily average. Ounces.
5 days of farinaceous diet . . .	372 ...	74
27 „ full diet	948 ...	35

Summary of Specific Gravity.

	Daily average.
5 days of farinaceous diet	1013
27 „ full diet	1020

SUMMARY OF QUANTITY OF ALBUMEN PASSED.

	Total quantity. Grains.	Daily average. Grains.
4 days of farinaceous diet . . .	354 ...	88·5
27 „ full diet	4576 ...	169·0

SUMMARY OF QUANTITY OF UREA PASSED.

	Total quantity. Grains.	Daily average. Grains.
4 days of farinaceous diet . . .	1529 ...	382
27 „ full diet	8163 ...	302

CASE 10. *Chronic parenchymatous nephritis.*—Michael W—, æt. 49, admitted into Guy's Hospital January 14th, 1891. Clinical clerk, Mr. A. M. Daniel.

He has had syphilis. Three months ago his feet and legs began to swell, and his urine became scanty.

On admission.—The only symptoms were œdema, the alterations in the urine, and some weakness. The liver and spleen could not be felt. He said he always felt much better on full diet than on milk or farinaceous. He left the hospital on February 13th. The œdema was less than on admission.

Date.	No. of oz. in 24 hrs.	Sp. gr.	ALBUMEN.		UREA.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
Jan.							
15	—	1012	3·25	—	1·1	—	Farinaceous.
16	58	1014	4·0	102	1·0	255	
17	86	1014	1·0	37	·75	283	
18	—	—	—	—	—	—	Full diet.
19	72	1015	7·0	221	1·1	348	
20	68	1016	9·0	268	1·1	309	
21	72	1018	9·0	285	1·4	444	
22	48	1017	8·0	168	1·5	422	
23	60	1020	10·0	284	1·6	422	Milk only, 6 pints.
24	60	1015	7·0	184	1·4	367	
26	80	1011	5·0	186	1·2	422	
27	82	1011	4·0	124	1·3	469	
28	72	1011	5·0	158	1·3	411	
29	62	1015	6·0	168	1·6	436	
30	64	—	6·0	168	—	—	Full diet.

Date.	No. of oz. in 24 hrs.	Sp. gr.	Albumen.		Urea.		Diet.
			Parts per 1000.	Total grs. in 24 hrs.	Parts per 100.	Total grs. in 24 hrs.	
Feb.							
4	42	1015	7·0	135	1·5	290	Full diet.
6	40	1018	6·0	105	1·6	281	
7	70	1015	6·0	184	1·4	431	
9	40	1021	9·0	158	1·8	316	
10	60	1020	9·0	237	1·7	448	
11	44	1020	10·0	171	1·8	348	
12	40	1017	7·0	153	1·8	316	
13	36	1018	8·0	126	1·8	284	

Summary of Quantity of Urine passed.

		Total quantity. Ounces.	Daily average. Ounces.
2 days of	farinaceous diet	144	72
4 „	full diet	260	65
6 „	milk diet	416	69
9 „	full diet	456	50

Summary of Specific Gravity.

		Daily average.
3 days of	farinaceous diet	1013·3
4 „	full diet	1018·5
6 „	milk diet	1013·8
8 „	full diet	1018·0

Summary of Quantity of Albumen passed.

		Total quantity. Grains.	Daily average. Grains.
2 days of	farinaceous diet	139	69
4 „	full diet	942	271
6 „	milk diet	1099	183
9 „	full diet	1437	159

Summary of Quantity of Urea passed.

		Total quantity. Grains.	Daily average. Grains.
2 days of	farinaceous diet	538	269
4 „	full diet	1523	380
6 „	milk diet	2527	421
8 „	full diet	2714	339

(For report of the discussion on this paper, see 'Proceedings,' New Series, vol. v, p. 103.)

LARGE CYSTIC MYOMA OF UTERUS

OF OVER TWELVE YEARS' DURATION REMOVED BY ENUCLEATION; RECOVERY.

WITH NOTES ON CYSTIC "FIBROIDS."

BY

ALBAN DORAN, F.R.C.S.,

SURGEON TO THE SAMARITAN FREE HOSPITAL.

Received January 25th—Read May 23rd, 1893.

E. R—, æt. 47, was admitted into my ward in the Samaritan Hospital on June 16th, 1892, suffering from a large abdominal tumour.

She was the mother of six children, the youngest being ten years old. About twelve or fourteen years ago she noticed a swelling in the right iliac fossa. In 1882 she consulted Dr. Ilott of Bromley, who sent her to me. There was a swelling on the right side of the uterus, feeling more like a dilated tube than a fibroid. Unfortunately the original full notes have not been preserved. I saw her frequently for two months till she ceased to attend, owing to the illness of her husband. She nursed him for over two years, then he died, and she worked hard in a laundry until the spring of 1891, when she was laid up

for a week with severe pains in the abdomen and lumbar region. The abdomen was then much distended. She could no longer work in the laundry, but was able to sit up and ply her needle till admission, when her health was failing rapidly.

She was rather tall and somewhat emaciated. She lay on her back in bed, feeling uneasy when sitting up or lying on one side. The abdomen was extremely distended, especially towards the left; the superficial veins were dilated, the integuments free from œdema. The girth at the umbilical level was 44½ inches; the distance from the ensiform cartilage to the umbilicus 11 inches; from the umbilicus to the symphysis pubis 14½ inches. The distension was due to a tumour which reached to and pushed forward the ensiform cartilage. There was resonance in the left loin, but not in the right. The lower part of the tumour was very prominent and fluctuated separately from the rest, from which it appeared to be divided by a deep groove. Over the upper and lateral parts was distinct fluctuation.

On vaginal examination the cervix could be felt rather high up and far back in the pelvis. The sound passed three inches forward and to the right; it could be moved somewhat forwards, but in no other direction. The tumour descended into Douglas's pouch and the left fornix, and on the other hand extended anteriorly downwards in front of the sound. A mucous polypus, about one inch in length, projected from the os.

The period, regular till a year before admission, had not been seen for six months. The average daily excretion of urine, independent of what passed during defæcation, was 35 ounces during the first week in hospital, the sp. gr. 1020. There was a trace of albumen. The temperature was normal; the pulse 100, hard, small volume.

The diagnosis was very uncertain; there had been no menorrhagia, and I have seen many large ovarian tumours of ten or twelve years' standing present similar features, physical and clinical. Hence some of my colleagues,

as well as myself, were inclined to consider that the tumour was ovarian, although before beginning the operation I expressed doubts, owing to the long history of a tumour.

As the bulk of the fluid caused great distress, I tapped the lower part of the tumour with the aspirator on June 20th, but only 6 pts. 17 oz. of a yellow, serous, imperfectly-coagulable fluid escaped. On tapping the upper part nothing came away excepting a little blood. I will presently return to this question of paracentesis. Though so little was removed good results followed in this case. The patient ate and drank better and felt in better spirits.

Five days later, June 25th, 1892, I operated, assisted by Mr. Butler-Smythe. I made a long incision ending four or five inches above the pubes, lest the bladder should be wounded. The surface of the prominent lower part of the tumour was exposed. It was pale, but not bright and shiny as in an ordinary ovarian cyst free from complications. The trocar was introduced, and twenty-five pints of orange-red fluid escaped. I was repeatedly obliged to introduce my hand into the tumour in order to tear off and clear away masses of broken-down tissue. On passing my hand upwards I was able to make sure that the upper tumour (which fluctuated separately) was continuous with the lower. I enlarged the incision, and then found that the cæcum and vermiform appendix lay on the surface of the tumour high up to the right, close to the liver. A tedious enucleation had to be undertaken, for the cæcum was not merely adherent, but lay on the peritoneum, which invested the tumour, forming a capsule. The elastic ligature was passed round the root of the entire mass. The uterus, containing several interstitial fibroids, was discovered, displaced to the left. The right and left appendages ran over the tumour, which had pushed up the pelvic and iliac portions of the peritoneum on the right side, hence displacing the cæcum. The catheter was then passed by Mr. Malcolm, who was present, but the fundus of the bladder did not rise high. The right round ligament looked like a ureter. It was not only stretched to the

extent of about six inches, but also much hypertrophied. It ran over the capsule round the right side of the tumour, and behind it to its normal insertion on the uterus. I passed the serre-nœud above the elastic ligature, then introduced two pins and cut the entire tumour away. The cæcum and appendix lay close to the serre-nœud wire. The elastic ligature was removed. I carefully sewed the parietal peritoneum to the serous lining of the stump on the proximal side of the wire. The rest of the wound was closed in the usual way with silkworm-gut sutures.

Even when removed, the relations of the tumour to the natural structures which had to be sacrificed with it were not to be detected at a glance. I have endeavoured to make the matter clear by the annexed sketch.

The fundus (*f*), distinguishable by its relation to the tubes, lay level with the surface of the tumour. It measured over 3 inches between the origins of the tubes. From its posterior aspect rose an interstitial fibroid (*a*), becoming subperitoneal, about 2½ inches in diameter at the base and 2 inches in vertical measurement. To its left was a smaller fibroid (*b*), growing from the left side of the uterus, and beginning to burrow between the layers of the broad ligament. The corresponding round ligament (*l. r. l.*) seemed, as it were, to spring from the smaller fibroid. The left Fallopian tube (*l. F. t.*), unobstructed, was elongated, measuring over 4 inches. Both layers of the mesosalpinx, that is the broad ligament above the level of the ovary, were in normal apposition, except close to the uterus, where they were parted to a certain extent by the smaller fibroid, *b*. That fibroid had burrowed into the broad ligament below the level of the mesosalpinx. The left ovary was atrophied and very flat, as the small fibroids *a* and *b* had jammed it against the bony pelvis. It contained two small atrophied follicles. The ovarian ligament was one inch long and very thin.

The relation of the small fibroids to the normal parts

on the left of the middle line is instructive, as it explains the relations of the great tumour that had developed on the right side. Had *a* and *b* grown as large as that tumour, the posterior reflection of the pelvic peritoneum would have been pushed upwards to the left hypochondrium, carrying with it the large intestine. The left

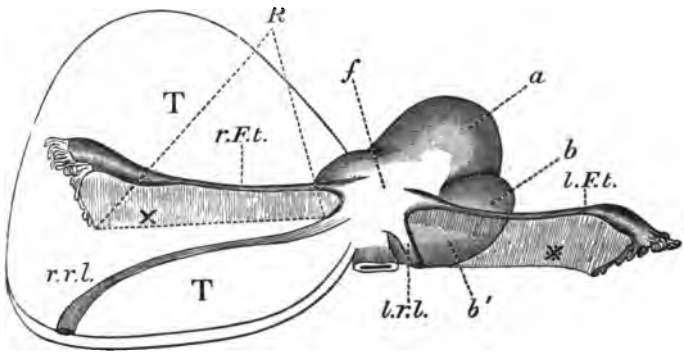

Diagram showing the parts removed with the cystic fibroid tumour.— T. Portion of surface of tumour. *r. F. t.* Right Fallopian tube. ×. Position of right ovary behind broad ligament. R. Line of reflection of broad ligament on to surface of tumour. *r. r. l.* Right round ligament, greatly elongated. *f.* Fundus uteri. *a.* Small fibroid. *b b'.* Another small fibroid, beginning to open up the broad ligament below mesosalpinx. *l. r. l.* Left round ligament. *l. F. t.* Left Fallopian tube. *. Position of left ovary behind broad ligament.

round ligament would have been greatly stretched by that portion of the tumour which pressed forward. There was no cystic degeneration in the fibroids on the left side; that change, so marked on the right side, must have greatly aided in displacing peritoneal relations.

The front of the fundus passed on to the right or main tumour. The right round ligament (*r. r. l.*) measured 9 inches as it lay on the serous capsule torn off the tumour. The uterine cavity lay entirely behind the tumour, and measured 3 inches vertically.[1]

[1] This measurement is of little value, as it is impossible to make sure how much of the uterus, above the cervix, remains behind.

The relations of the right tube and ovary were remarkable. The Fallopian tube (*r. F. t.*) was 5½ inches long and unobstructed; the right ovary 3¼ inches long, and ½ inch in vertical measurement. It bore an oval cystic cavity full of recent clot, and one inch in long diameter. The ovarian ligament was only half an inch long. The noteworthy feature was that, notwithstanding the enormous size of the right tumour, and the extent to which it had displaced the peritoneum elsewhere, the right mesosalpinx was almost normal. In the sketch, R represents the line of reflection of the layers of the broad ligament on to the tumour. In fact, the tube was attached to the tumour by its mesosalpinx. In a broad ligament cyst, and in a burrowing ovarian cyst, the tube always lies in contact with the tumour which effaces the mesosalpinx by burrowing between its layers.

In this case the tumour developed on the right side of the uterus (like *b* on the opposite side), and burrowed between the layers of the right broad ligament below the level of the mesosalpinx, finally dissecting up and distending the pelvic peritoneum, stretching the round ligament in front, and carrying upwards the cæcum. The distending and stretching of the peritoneum must have prevailed over the dissecting-up process, else the tumour would have lain against the parietes entirely in front of the parietal peritoneum, or else against the aorta and posterior boundaries of the abdomen behind the posterior reflection of the peritoneum. As it was, a deep peritoneal pouch lay between the tumour and the structures in front of and behind it. Stretching (with hypertrophy, of course) of the peritoneum is seen in broad ligament cysts which may be of great size, although the relations of the peritoneum around them remain absolutely unchanged. Here it is the mesosalpinx alone, normally a very small peritoneal fold, that undergoes so much distension. In my case the tumour stretched the peritoneum forming the broad ligament below the level of the ovary, and also stretched part of the peri-

toneal lining of the right iliac fossa. As usual, when it began to stretch, it ceased to dissect up and displace any more peritoneum.

The solid part of the tumour weighed 10 lbs. 6 oz.; the fluid amounted to 25 pints. Hence, including the 6 pints 17 oz. removed five days previously, the patient was relieved of about 42 lbs. of solid and fluid material. The tumour itself was divided into two parts by an hour-glass constriction. Each part consisted of a main cavity, communicating with the same in the other half, as I mentioned in describing the operation, and bounded by a solid wall which varied in thickness from one eighth of an inch to over two inches. The solid matter was throughout, wherever cut into, a spongy tissue, as in soft uterine fibroids. It was full of cavities, which gave it a bullous appearance on section. A piece, where this appearance was most distinct, was preserved for microscopic examination. From these solid walls much broken-down material projected into the cavity. Beyond what had been torn down in the course of the operation, there were masses of pedunculated bodies, two or three inches long, apparently fibrinous. They may represent changes in old coagula, such as are seen in hydroceles and bursal sacs. Mr. Targett made several sections of the "bullous" tissue,[1] described above, in the laboratories of the College of Surgeons. The tissue consisted of loose bundles of almost pure plain muscle-fibres in a colourless matrix; there were spaces which bore no lining. Hence the histology of the tumour throws no light on its origin. I

[1] Since this paper was prepared, M. Maurice Cazin examined a cystic fibroid which M. Segond had removed by operation; it was found attached to the fundus by a broad pedicle. The greater part of the cystic structure was due to œdematous softening; but near the surface of the tumour were minute cysts, not larger than millet-seeds, which were found to be lined with cylindrical epithelium. The uterine mucous membrane, though so far from the periphery of the tumour, seemed to M. Cazin to be the only possible source of origin of these epithelial cysts. Possibly, in my own case, the bullous structures might have originally been lined with epithelium which had become shed through a degenerative process.

believe that the cavities which appeared like bullæ on section might have been developed from lymphatic vessels. The endothelial lining of these vessels in morbid growths is not easy to detect by the microscope.

In the after-treatment of the case the stump gave me some trouble, as, notwithstanding every precaution, dressing with absorbent gauze, tannin, and iodoform, there was a considerable amount of sloughing below the level of the wire during the second week. The sloughy tissues were gradually trimmed away, and by the seventeenth day the wound was clean and granulated freely. It was packed with iodoform-gauze, and washed out daily with red lotion. The patient was discharged on August 9th.

When I saw her last, on April 28th, 1893, she was in excellent health and had gained flesh. The cicatrix, deeply puckered, was firm and healthy; there was no indication of any hernial protrusion. It is remarkable that a slight "show" of blood occasionally issued from the vagina, though the appendages had been entirely removed, and the greater part of the uterus amputated, the stump undergoing extensive destructive changes during convalescence. The show is probably due to the small mucous polypus.

I did not remove the mucous polypus, as it gave no trouble; there was neither discharge nor hæmorrhage. I know of a case where a polypus of this kind was twisted off, a large uterine fibroid existing. The removal of the fibroid was to have taken place a few days later, but the "minor" operation killed the patient.[1]

It cannot be said that the pathology of fibro-cystic myoma of the uterus is thoroughly known. According to some authorities the cysts are formed by breaking down of myomatous tissue through œdema or fatty degeneration, but the manner in which this breaking down begins has been explained on different hypotheses. Certain pathologists believe that the cysts are originally developed from dilated vessels or blood-sinuses. Others

[1] See my 'Handbook of Gynæcological Operations,' p. 315.

maintain that they represent obstructed lymph-channels. In the later stages all these changes may be detected, but the initial lesion cannot then be determined.

Virchow distinguishes a myxo-myoma from simple œdema. Yet may not the latter be the cause of myxomatous degeneration? Œdema and softening, Gusserow admits, may result from changes during pregnancy. As damage to the nutrition of a solid "fibroid" during parturition may cause its delivery (if submucous) or suppuration or gangrene, so similar damage may set up cystic changes. My patient had borne children since the tumour had grown large enough to attract her attention. The disappearance of myoma during pregnancy may be explained, Gusserow suggests, in the same manner. W. Müller, among recent writers, does not reject œdema, but his views on lymphangiectasis will presently be considered. The myxomatous degeneration and the condition resembling "molluscum fibrosum" of Virchow, which have been observed by Bland Sutton and others in specimens examined under the microscope, no doubt exist in the solid matter around fibro-cysts, but these authorities do not prove that they cause the cysts to develop.

Dr. Friedrich Uter has prepared one of the most recent monographs on the development of cysts by breaking down of tissue. He and Dr. Schaumberg detected areas of necrosed myomatous tissue. He describes the appearances with great care. The specimens were from a case of fibroid impacted in the pelvis. The patient suffered from severe local symptoms with peritonitis and fever. But the "fibroid" apparently contained but one cyst, "about the size of a cherry;" it was almost filled by a piece of necrosed tissue which was attached by a pedicle to the bounding wall. That wall was irregular and bore no epithelium. No myxomatous degeneration was noted. Numerous small circumscribed deep red patches were seen scattered over the cut surface of the solid matter, and these, Dr. Uter believes, represented the origin of the cyst. But impacted fibroid is an extreme condition,

and there is no evidence that in my case or in many other cases of cystic fibroid impaction ever occurred. Dr. Uter admits the existence of other forms of cystic uterine tumour.

The theory that the cysts develop from pre-existing cavities must now be considered. When no epithelial lining is detected in a large or even a small cyst it does not follow that the cyst arose from a tissue devoid of epithelium. Indeed, the two structures, blood-vessel or lymph-channel, whence it is said the cysts arise, bear endo- not epi-thelium, and endothelium does not readily adapt itself to the progressive changes which epithelium undergoes in adenoma and more malignant growths.

Virchow's myoma telangiectodes is a distinct form, at least anatomically, though pathologically the distinction is less marked, and clinically there is none at all. So thinks Gusserow, who compares the cavernous tissue to the corpus cavernosum penis, and notes that the tumour often increases perceptibly in size at the menstrual period. This change is often seen in solid fibroids. He notes Leopold's case where the cystic cavities were full of blood. This appearance is not seen, as a rule, in the majority of fibroids with large cysts.

The evidence that Virchow's and Leopold's tumours represent a distinct and rare class seems strong though not conclusive. On the other hand, there is good reason to believe that a large proportion of cysts in fibroids are in origin lymphangiectases. Pozzi speaks of their endothelial lining as distinctive, but I have already noted that there is no evidence that the endothelium may not disappear when the cyst grows large. W. Müller, who has examined numerous myomata with large, medium, and minute cysts, believes that lymphangiectasis is far more common than is usually supposed. Many cases have never been submitted to microscopic examination. Simple œdema must be distinguished from vesicular œdema ("blasige Œdem"—Virchow) the latter being due to dilated lymph-channels. There are, Dr. Müller ob-

serves, evident intermediate stages between the two forms, representing incipient lymphangiectasis. He insists that a tumour of this kind is in no sense a lymphangioma.

I am strongly inclined to agree with Dr. Müller, for I know from long clinical experience that in large fibroids there is evidence of great obstruction to the lymphatics. Large yellow bubbles are often seen in the broad ligaments during operation, whilst enormously dilated lymphatics run over omentum adherent to the fibroid. The bubbles are thin-walled spaces (lacunar cysts of Verneuil), and lymph drains from them after the removal of the tumour, so that they disappear. In these cases I have traced dilated lymphatics entering the fibroid.

The fluid in the cysts bears the characters of lymph. It is rarely like the altered blood seen in old hydroceles and in the loculi of ovarian cysts. Moreover, when real blood is found in a cyst in a "fibroid," it may have escaped from ruptured vessels in the degenerate cyst-wall; it does not prove that the cyst was originally a blood-vessel. As a rule, says Gusserow, the fluid is pale yellow and coagulates imperfectly. Heer, it is true, found that in only eleven out of seventy cases did this spontaneous coagulation take place. Keith, in his first case, notes how the red serous fluid turned into a jelly in the course of the operation. In my own case the fluid might well have originated from lymphatics, without any admixture with blood.

There is reason to suppose that treatment by electricity may at least encourage cystic degeneration of "fibroids." Dr. Bache Emmet removed a large cystic fibroid, with the uterus entire, from a woman aged forty. Ten years before operation it was as large as an egg. Eight years later it was about the size of a uterus at term. It was treated by galvano-puncture, powerful currents being employed. There was temporary relief, then in a year menorrhagia set in, and the tumour increased greatly in size. Milder galvanic currents, without puncture, were used in vain, so the tumour was removed. Dr. Harry Sims, in

reference to Dr. Bache Emmet's case, observes that in one case upon which he had operated he found that no reduction followed the electrical treatment. The tumour was honeycombed throughout by large disintegrated masses. Mr. Knowsley Thornton observed cystic degeneration in a case where the abdominal wall was freely scarred by previous "Apostoli's treatment." In these cases necrosis, as described by Uter, possibly occurred. In none is there distinct proof that the electrical treatment actually caused the cysts to develop.

From pathology we will now pass to diagnosis. Many writers testify that cystic fibroids are often first detected in the course of an intended "ovariotomy." This had happened to experienced authorities such as Keith. In Case 1 "no very careful examination was made, for now I never doubted that the tumour was ovarian." In Case 5 "a single, almost momentary examination, was made, and the opinion given was that there was no doubt that the tumour was ovarian." In Dr. Keith's remarkable case (36) he hesitated, for given reasons, between "uterine fibrous cyst" and "some undescribed form of retro-peritoneal tumour." Case 40, still more extraordinary, was mistaken by two distinguished Italian professors for an ovarian tumour, but Dr. Keith made a correct diagnosis.

Gusserow relates a case in Spiegelberg's practice, where a cystic fibroid was tapped by mistake, being taken for an ovarian cyst. In Cushing's case, "the tumour when exposed looked like a multilocular ovarian cyst." In my case, though the surface of the cyst was not bright and shiny, as in a multilocular ovarian tumour free from adhesions and inflammatory or degenerative changes, it was not unlike many such tumours subject to these complications. The reddish capsule of a broad ligament cyst resembles uterine tissue. In cases of my own, and also in others under the care of my colleagues, I have seen the surface of a uterine fibroid appear white and shiny when exposed. Thornton, McMurty of Louisville, Kollock, and Henry Byford relate remarkable errors of diagnosis.

In several of the cases above quoted, as in many others where cystic fibroid of the uterus was taken for an ovarian cyst, the tumour was pedunculated. Hence the uterus moved freely from it, and hence it is very natural that it was mistaken for an ovarian tumour. Sir Spencer Wells notices errors of this kind in his well-known writings. Gusserow observes that cystic fibroids are said to grow quickly, whilst ovarian cysts grow slowly, "but numerous exceptions on both sides are sufficient to show that this point of diagnosis is unsafe." All observers of any experience will agree with him. Ovarian cysts often increase rapidly; on the other hand, when the vessels of the pedicle become gradually obliterated, they not rarely remain stationary (whether large or small) for many years. Some uterine cystic fibroids grow rapidly; some, as in my case, increase slowly.

The catamenial history is often misleading; menorrhagia may be present when the cyst is ovarian, and cystic fibroids are seldom associated with that condition. In my case no "show" had been seen for six months, yet there was a mucous polypus, which often keeps up hæmorrhage. The menopause is frequently deferred till fifty or later in patients subject to fibroid of the uterus.

The fluid found in large cystic fibroids has been described. It is certainly different from the characteristic glairy fluid which fills the cavities of multilocular ovarian tumours or the watery contents of broad ligament cysts. But tapping is dangerous, and not to be advocated in doubtful cases as a means of diagnosis. Again, fluids in cysts may undergo many changes which alter their appearance and puzzle the observer. Large ovarian cysts not rarely contain a lymph-like, pale yellow fluid.

The relations of cystic uterine fibroids are often puzzling. I have just referred to the close resemblance of pedunculated cystic fibroids to pedunculated ovarian cysts, as far as physical signs can guide us. Both these tumours may, on the other hand, be sessile. Diagnosis then becomes particularly difficult. The uterus in both cases

appears more or less intimately connected with the tumour. The sound may fail to settle the difficulty. The uterine cavity is not always enlarged in cystic fibroid disease, whilst in cases of large sessile ovarian tumours it may be elongated. Of course careful employment of the sound for ascertaining whether the uterine cavity lies in front of the tumour or behind it is necessary for diagnosis. It must not, however, be forgotten that the sound is a dangerous instrument in many cases of uterine disease, especially cystic fibroid. Thus in Spiegelberg's case, already quoted, where the cyst was tapped by mistake, the patient died of septicæmia from suppuration of the tumour due to perforation of the uterus by the sound. On two occasions that instrument was made to pass 6⅞ inches into the mass.

Dr. Lediard, of Carlisle, relates an interesting case of "fibro-cystic myoma of the uterus," where the patient was admitted into hospital for free hæmorrhage and vomiting. The sound passed 4 inches into the uterus. The symptoms continued, and a month after admission the temperature rose to 103·6°. Nine days later the sound was passed both into the uterus and the bladder; no bad symptoms followed, but at the end of a week the sound was introduced into the uterus and the bladder examined as before. There was evidence of decomposing retained discharge. On the following day the patient was restless, hot, and sick. Symptoms of septicæmia set in. The patient died on the ninth day after the last sounding. There was no trace of abrasion within the uterus, but no examination of the veins was made. The lungs were œdematous and congested; the kidneys contained millet-seed collections of pus. Dr. Lediard gravely suspected the sound as the cause of the fatal result. Dr. Gervis, when the case was read before the Obstetrical Society, expressed his fear that Dr. Lediard was not alone in his experience of the occasional mischief which followed the most careful use of the sound. A very slight abrasion might be followed by absorption if any septic products

were at hand. I have known death to occur within a few hours after careful passage of the sound in a case of malignant tumour of the ovary. The penetrative power of the sound, in some cases where the uterus is apparently healthy, is well known. Little harm is done in such cases, but, as the above records show, it is quite otherwise when a cystic fibroid exists.

In short, diagnosis between cystic uterine fibroid and ovarian cyst is sometimes barely possible, and two of the favourite methods of diagnosis—tapping and the sound—are neither sure nor safe.

Whilst some cystic uterine fibroids have been taken for ovarian tumours, others have been taken for solid uterine fibroids. When the walls of the tumour are thick this error is natural, for fluctuation is often obscure or absent. A soft, œdematous, solid fibroid often gives to the hand a sense of fluctuation. Sudden increase of growth is a frequent symptom when cystic degeneration of a solid fibroid has set in, but it is not invariably seen. In my case the increase was very slow and very uniform. The presence or absence of menorrhagia is in no way reliable as a symptom. Pedunculated cystic fibroids do not cause menorrhagia, but they may be complicated by small submucous growths or mucous polypi, and then there will be more or less bloody discharge.

I have mentioned that a mucous polypus grew from the cervix in this case. Matthews Duncan notes that "a fibroid often induces the growth of mucous polypi of the body or of the neck of the womb, and these aggravate and prolong hæmorrhage." How a fibroid can "induce" this new growth he does not explain. In my case, as already noted, the polypus neither caused hæmorrhage nor deferred the menopause.

The consideration of the treatment of cystic fibroid is a matter of high importance. The presence of cysts is a distinct complication, and when the tumour is mainly a single large cyst, as in my case, an operation is often needed, the more especially as the tumour does not tend

to atrophy at the menopause, and the pressure-effects increase in severity.

When the nature of the disease is almost certain, I doubt that any surgeon would recommend tapping. In cases of ovarian cysts, where there is a great amount of fluid with marked dyspnœa, the patient being old, tapping is sometimes advisable. The sudden removal of a great quantity of fluid during operation may cause serious pulmonary complications—a grave matter when from other causes there is much shock. Hence tapping before operation allows the patient time to recover the perfect use of her lungs and improve digestion, whereby she is the more fit for ovariotomy. In my own experience excellent results have followed this practice. In my case of cystic fibroid this improvement actually took place. Yet had I been more certain, I should not have tapped. Experience shows that in cystic uterine fibroid the practice is very dangerous. Gusserow remarks that, according to Leopold and Fehling ten out of eleven cases of systematic tapping proved fatal. The rigidity of the uterine cysts allows of the entrance of air, and large vessels or very vascular layers of tissue are often wounded. Not rarely the trocar is hard to drive into the cavity of the cyst, and only a few drops of bloody fluid escape. The soft, œdematous, breaking-down tissue bordering on the cyst may be pushed forwards and obstruct the cannula. In Dr. Keith's first case, when the tumour was already exposed at the operation, "there was a difficulty in getting in the trocar. The cyst was quite thin, but it required a very strong effort to push in the instrument." In Case 5, during operation, much force was required to pierce the tumour, and then nothing but a little blood escaped. Should the tumour be a very soft, solid uterine "fibroid," which often feels cystic, tapping would involve great dangers.

An exploratory incision is but a step towards operation. In my own case, as in many others, the mere exposure of the tumour by no means settled the diagnosis. This subject, the appearance of the cyst-wall, has, however,

been already discussed. In Sir Spencer Wells's case, performed at Dublin in 1864, the nature of the cyst was not evident at a glance even after exposure and tapping. The tumour must be manually explored with great caution, so that its relations may be determined.

There can be no doubt that, other conditions being the same, the removal of the tumour is more justifiable and more necessary when it is cystic than when it is solid. Some of the reasons for this opinion have already been given. Gusserow shows, from the collected evidence of earlier workers, that the duration of cystic fibroids is relatively short, the longest period being ten years, whilst in many cases death occurred within a few months. It is not possible to determine how long the tumour had become cystic in my case. Suppuration through thrombosis in their substance or neighbourhood is frequent. The rapidity of growth, and tendency to suppuration and to increase instead of diminish at the menopause, all suggest that an operation is needed.

When the tumour is pedunculated, as is so frequently the case, its removal is not very difficult. The pedicle is harder to secure than that of an ovarian tumour. The ligature may slip, and the serre-nœud involves sloughing which may extend into the uterus or give rise to troublesome hæmorrhage. When the cyst burrows under or behind the parietal peritoneum, or in front of it, the difficulties of the operation are great, as my case proved. The complications may be very intricate, too varied and complex for any description here in detail. Fuller information will be found in the writings of Sir Spencer Wells, Keith, Pozzi, and others. To the special difficulties arising from displacement of peritoneum may be added those not rare in cases of solid "fibroid"—such as vascular adhesions and difficulty in making a pedicle. In a case where Dr. Cushing successfully operated, the patient, aged fifty, suffered severely from dyspnœa owing to the bulk of the tumour. She had to be made to sit crosswise on the table. As the tumour was rolled out

the veins, relieved from pressure, began to distend, and syncope threatened to come on; she was placed in the ordinary recumbent posture and the operation completed. Other operators have experienced great trouble owing to rents in the peritoneum, which required union by suture, unless the edges of the serous membrane could not be brought together, and then the raw surface required drainage.

The uterine tissue is often œdematous and otherwise degenerate. Hence the stump requires very careful dressing, and in spite of all precautions may, as in my case, cause much trouble during the after-treatment. Perhaps firm packing with absorbent gauze, frequently changed, is the best kind of dressing. It is of particular importance that the peritoneum be well united by sutures to the stump on the proximal side of the serre-nœud wire. This practice prevents retraction of the stump into the peritoneal cavity, an accident always serious in hysterectomy, and especially dangerous when the remains of the amputated uterus are soft and bulky, as in cystic fibroid disease, so that the sloughing process may extend some distance below the level of the wire.

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(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. v, p. 114.)

ON

ENTERORHAPHY BY INVAGINATION

(MAUNSELL'S OPERATION),

WITH A CASE OF ARTIFICIAL ANUS TREATED BY THIS METHOD.

BY

STANLEY BOYD.

Received February 11th—Read May 23rd, 1893.

As Maunsell's method of performing enterorhaphy may not be familiar to all present, a short account of it will not be out of place. It is not one of the many recent plans which have resulted from the activity excited in this department of surgery by the publication of Senn's valuable and ingenious work. It is more than ten years since Dr. Maunsell, then Lecturer on Surgery in the University of Otago, New Zealand, thought out the details of the method and proved its efficacy, so far as was possible, upon the cadaver. Not until December, 1886, did an opportunity of trying the method upon the living subject occur; then Maunsell performed it upon a child suffering from complete obstruction, due to cancer involving some 4 inches of the small bowel. The resection of this mass and suture of the bowel-ends occupied only

half an hour. The child was much exhausted at the time of operation, and died without obvious cause on the sixth day. The bowel-ends were united, and there had been no leakage.

An account of the method and of the above case was read at the Medical Congress at Adelaide in 1887; but, so far as I have been able to discover, Dr. Maunsell has had no imitators.

The following are the steps of Maunsell's method of performing enterectomy and subsequent enterorhaphy:— (1) Clamps are applied above and below the piece of bowel to be excised. The clamps used are large safety-pins, on to the bent portion of which bits of sponge, just large enough, when moist, to fill up the interval between the fixed and movable parts of the closed pin; thus armed the pins are passed through the mesentery and fastened round the bowel, compressing it gently. (2) The excision having been performed and the bleeding points in the mesentery dealt with, the ends are approximated by two long horsehair sutures passed through the mesenteric and free borders and tied within the bowel (Fig. 1); both

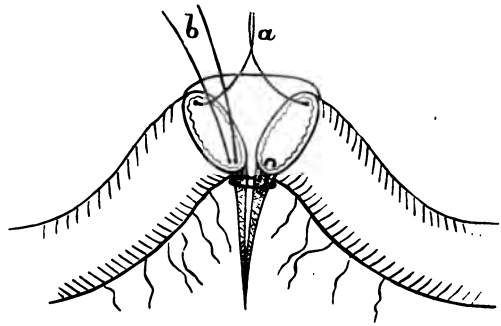


FIG. 1.—Sutures of approximation in free (*a*) and mesenteric (*b*) borders passed, but neither tied.

of these sutures start and end on the mucous surface, and the first is so introduced as to completely close both the mesenteric triangles, and, when tied, to bring together

the outer aspects of the bowel ends covered by peritoneum. (3) In the free border of one end—the larger if there is any difference between them—and 1 inch from the line of section, an incision 1½ inches long is made; the ends of the two sutures just introduced are passed out through this opening (Fig. 2) and gently drawn upon; an invagination

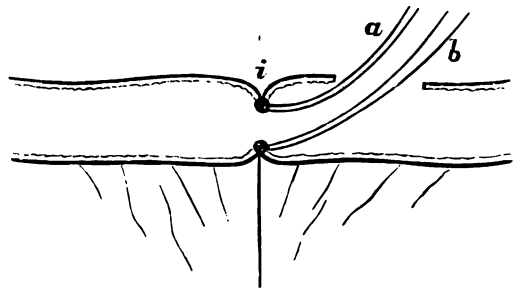


FIG. 2.—Sutures of approximation tied, and their ends (*a* and *b*) passed through a cut in the free margin. *i*. Line of section.

of one end into the other is thus produced, its free end projecting through the cut in the unattached border and the serous surfaces being in contact (Fig. 3). An assistant

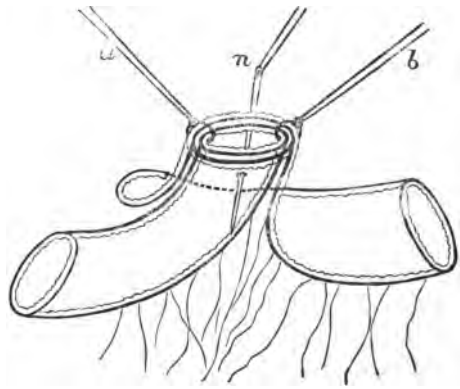


FIG. 3.—Traction has been made on *a* and *b*, producing an invagination, the layers of which are visible (dark lines = serosa, wavy = mucosa). *n*. A needle passed through all four layers of bowel.

keeps up gentle traction on the sutures, and thus draws the lumen of the invagination into a long oval form, one side of which is held towards the surgeon. (4) Fine, straight, round sewing needles, threaded with horsehair, are passed through the whole thickness of all four layers of intestinal wall—across the lumen of the invagination—and about a quarter of an inch from their cut edges (Fig. 3). After each of these sutures has been passed, the horsehair is to be drawn up with forceps where it crosses the lumen, divided, and the halves tied as quickly as possible all round the circle of the cut edges. (5) All long ends are cut short, the knots being within the bowel, and the intussusception is reduced. (6) Finally, the longitudinal wound in the free border is closed with a continuous horsehair suture not passing through the mucosa (Fig. 4). (7) The clamps are removed, the protruding bowel is carefully cleansed and returned to the abdomen.

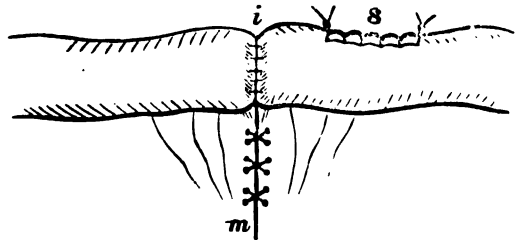


FIG. 4.—Invagination reduced. *i*. Line of union; stitches too evident. *s*. Wound in free margin closed by continuous suture. *m*. Mesenteric V closed by interrupted stitches.

A junction of this kind made in dead intestine will be found to bear a very considerable water-pressure without leaking; it is quickly effected, no extraordinary apparatus is required, no plates, no tubes, which may or may not fit, and which are almost certain not to be at hand when wanted; it may, indeed, be said that the instruments necessary to carry through this operation may be found

in every household. The mesenteric border seems to be securely dealt with; apertures of different size are readily united. The idea of making an incision in one end of the divided bowel and drawing out through it, back to back, the edges to be united, was derived, Dr. Maunsell has told me, from seeing a tailor unpick the lining of a sleeve in order to mend a hole in the seam between the sleeve and body of a coat. It appears to be well worth bearing in mind, as being capable of wide application. Naturally, Maunsell has extended his method of enterectomy and enterorhaphy to the treatment of irreducible intussusception, and A. E. Barker was led independently to adopt a very similar plan; F. T. Paul has recently published, with acknowledgments to Dr. Maunsell, a plan of effecting a communication between the duodenum or jejunum and the posterior surface of the stomach by operating through an incision, to be subsequently closed in the anterior surface of the stomach; and it is likely that the easiest way to the posterior surface of the stomach for the purpose of suturing a perforation or excising an ulcer and suturing the gap would be through the anterior wall.

The points against Maunsell's method seem to be the extra longitudinal incision and the passage of the sutures through all the coats of the bowel. Experience alone can decide the value of the method, but it may be pointed out that it is easy, if there is any doubt about the reliability of the union, to add a few Lembert's stitches in the neighbourhood of the mesenteric border or elsewhere, to render the invagination irreducible by a few stitches round its neck, or to apply an omental graft round the junction and thus render it more secure.

I will now turn to the case in which Maunsell's operation was tried.

Mrs. E. H—, a healthy, well-nourished woman æt. 36, was admitted into the Charing Cross Hospital under my care on November 15th, 1892. She had had a right femoral hernia for about four years; after unusual efforts it

had sometimes enlarged and become painful, and sometimes it had remained down for a week at a time. But it had never been strangulated until April 28th, 1892, when being in bed and not straining in any way she was seized with pain in the abdomen, vomiting, and constipation. Dr. W. Griffith, of West Angle, to whom I am indebted for these notes, saw Mrs. H— on April 30th, and found a large right femoral hernia, with a slight impulse on coughing, tender and covered by greatly swollen and œdematous tissues. He was unable to reduce it, so ordered Pil. Opii, gr. j, every four hours, and applied cold cloths to the swelling. Next day Dr. Griffith and Dr. Stamper found the patient better, vomiting having ceased. A. C. E. was administered; the swelling, still proving irreducible by taxis, was cut down upon and reduced without opening the sac. Everything went well, and milk was taken freely on May 2nd and 3rd, but on the night of May 3rd the patient vomited a large quantity of yellow fluid, and complained of pain in the right iliac region. On the 4th she was put upon nutrient enemata, the vomiting ceased, and she seemed to be going on well up to the 12th,[1] when a slight discharge began from the wound, which increased and became fæcal on the following day. The bowels acted *per viam naturalem* on the 24th, and motions passed this way twice or thrice a week up to the time of admission into hospital, some six months later. An enema was generally required to provoke these actions *per anum*, but the escape of more or less fluid fæces from the groin was practically constant, and took place at first from two openings, of which one, the more external, had closed before admission. The patient stated that abscesses had formed during the healing of the wound in the groin. It will be seen that many points in the above history are most singular, especially when taken in connection with the state of matters found at the operation.

On admission the patient was a healthy, well-nourished

[1] The patient is positive that fæces escaped from the wound on the 7th.

woman, who had lost little or no flesh. In the right groin, roughly parallel to Poupart's ligament, was a depressed and irregular scar, 2—3 inches long, ending internally over the saphenous opening. Through an aperture here, just admitting the forefinger, fluid fæces and mucus escaped, and a short prolapse of mucous membrane occurred. A finger introduced through the opening entered a considerable subcutaneous cavity running in towards the mons; from the deep aspect of this the little finger could be passed through the crural ring —of which the more rigid boundaries were easily recognised—into a large bowel cavity; neither spur nor ileo-cæcal valve could be felt. The skin of the thigh and abdomen round about the prolapse were excoriated for 2—3 inches, and were thick and rigid from inflammatory infiltration.

The balance of evidence seemed to be in favour of the diagnosis of "cæcal fistula." There was only one opening in the groin, and no spur could be felt; there was a history of a second opening, but this had probably been a sinus left by an abscess, and motions had been passed *per anum* up to admission. The points which gave rise to uncertainty were that, although the supposed opening in the cæcum was small, by far the greater part of the fæces came away through it; that the motion escaping was always more fluid than might be expected from the cæcum, and that the ileo-cæcal valve could not be felt.

On November 26th the patient was prepared for operation by the administration of Ol. Ricini, oz. ss, by the use of simple and salicylated enemata, by ordinary washing of the inguinal region, and by the application of wool wrung out of 1 : 1000 perchloride lotion over the area of the groin. As motion flowed continually from the wound up to the time of operation, cleansing was effected, after ether had been administered, by scrubbing the parts well with a nail-brush dipped in 1 : 20 carbolic brought up to the strength of 1 : 500 with solid perchloride. Irriga-

tion with warm iodine lotion was kept up throughout the operation.

The scar and fistula were now included between hemi-elliptical cuts, which were deepened on to the protruding bowel in the interior of which the left forefinger was kept. At first the knife passed through the densest scar tissue, but, once the bowel wall was reached, each touch of the blade caused the piece of gut external to the ring to come well down. Finally the ring was reached, and the adhesions—which, as usual, were limited to its immediate vicinity—were divided; in doing so, however, two small perforating wounds of the bowel below the ring were inflicted. There now slipped out of the abdomen, through the ring—not the cæcum, but an unmistakable piece of small gut, continuous with that which had been dissected from its adhesions outside the ring; evidently a loop of small gut had sloughed and separated, and this was the end of the proximal segment. The distal end was soon found, adherent to the proximal on its hinder and mesial aspect—its free extremity being puckered up as if a thread had been run round it and drawn tight; it was completely closed by scar tissue, but was easily separated from the proximal end. This closed distal end lay entirely above the ring; apparently it had never presented upon the surface, and the second opening described by the patient was a sinus mouth.

Both ends were now drawn well down into the thoroughly cleansed wound and laid upon guards; there was no need for any clamp, as discharge of fæces had ceased.

On examining the parts it was found that between the ends there was a free edge of mesentery one and a half to two inches long, corresponding to a loop of bowel which had sloughed. Nothing was said in the history of the separation of any piece of bowel, yet a considerable bit must have come away. Probably no intestinal contents passed from the lower to the upper segment of the bowel after the reduction of the strangulated loop. The two or three motions a week, passed since the herni-

otomy six months earlier, must have come from a well-filled lower bowel. The discovery of the true state of matters, however, led to more accurate inquiries upon the subject of these motions, and elicited that they had been quite small, loose, pale, and foul-smelling, but the woman had no doubt but that they were fæcal. She passed nothing *per anum* whilst in hospital.

To return to the operation—a piece of the proximal end of the bowel about two inches long was cut off with scissors, including all the part which had been adherent and a little more; and about half an inch of the distal end was similarly removed. The latter end was much smaller than the upper, and was gently dilated by the insertion of the forefinger up to its base. A wedge of mesentery was then excised, the vessels tied, and the edges brought together with fine catgut. One of these sutures passed through a vessel, the bleeding from which was controlled only by another stitch passed nearer the spine and further from the cut edge. The pared edges were united by Maunsell's method, but I departed from the above description in certain points—sometimes with advantage, sometimes with the reverse. The longitudinal incision was made in the *distal* end, and the upper, *larger* end was invaginated—with a little difficulty—into the lower *smaller* end, with the intention of fixing the invagination so as to cover the line of union should it prove weak. This plan was not adopted, for when the invagination was reduced a shallow groove marked the line of union, and the stitches could scarcely be seen crossing the bottom of it. To make doubly sure, two or three Lembert's stitches were added on each side of the mesenteric attachment. At first the finest silk was used for the enterorhaphy, but the wet ends were difficult to pick up and disentangle; horsehair, recommended by Maunsell, was found much pleasanter to work with, and is, no doubt, a safer material for a penetrating stitch.

Some of the needles were passed through all four

walls of the bowel ends and across the lumen of the intestine; others were passed through only two walls, and the sutures were tied as they were inserted. The latter plan was found to be the more rapid—so much time was lost in drawing up the loops from the lumen of the bowel and in selecting corresponding ends. Great care was necessary to ensure that the cut edges of the peritoneal coats were equally drawn up, and that each stitch passed a good quarter of an inch below them, for the mucous membrane prolapses and conceals the peritoneal edges, which are of chief importance. The longitudinal wound in the distal end was closed by *two* rows of continuous horsehair suture, and this bit of bowel—small at the start—was not of greater circumference, after suture, than an adult little finger at its base. After thorough cleansing, the sutured bowel was reduced into the abdomen by a few minutes of gentle pressure—so little was the size of the bowel increased or its flexibility diminished by this mode of suture. The femoral ring seemed to be of about normal size.

By way of "radical cure," three fishing-gut sutures were used to approximate the pectineal fascia and the inner end of Poupart's ligament; the infiltrated skin and subcutaneous tissue on either side of the wound were raised for some distance, so as to render possible union all along by a continuous suture. The wound and the excoriated skin around it were thickly plastered with a paste of iodoform powder in 1 : 1000 perchloride lotion, and a dressing of alembroth gauze and wool was applied.

The patient was put to bed with the right thigh flexed over a pillow. Flatus was passed *per anum* the same night, and she slept well. Beyond slight pain about the navel just after the operation she had no symptoms. *As to feeding:* at first she was allowed only teaspoonfuls of hot water by mouth; by rectum she was given 4 oz. of pancreatised milk gruel every four hours. These enemata were continued and retained for about a fortnight, though less and less nourishment was given in this way, while the

quantity of food given by mouth was steadily increased, and the frequency of administration was diminished. Solid Brand's essence was thus given on the 10th, three eggs and the lean of a chop were allowed on the 18th, fish on the 25th, and ordinary full diet on the 31st day. The *bowels* showing no tendency to act, a simple enema was given on the twelfth day without result, another on the fourteenth and daily afterwards. On the fifteenth day a copious fluid motion resulted, and on the sixteenth a large, soft, solid, pale yellow stool was passed; it was not offensive. No blood, slough, or other abnormal constituent was noticed in the stools. After the sixteenth a daily enema kept the bowels regular.

After operation the patient's temperature rose to 100·4° in eighteen hours, then fell gradually to normal on fifth day. The dressing was changed on this day, as it was feared that the wound and its vicinity might not have been perfectly disinfected; but everything seemed normal, and the wound appeared healed. All stitches were removed, and this was no doubt a mistake; for on the sixteenth, though quite sweet and dry, the wound was found to be gaping centrally (where the fistula had been excised), and at the bottom of the small cavity a clot was seen which slowly organised. Not until the fortieth day was healing perfect; the patient had been sitting up previously, and she was now allowed to walk. She left the hospital on January 11th (forty-seventh day) in very good health, eating ordinary food and with regularly acting bowels. When last seen, on January 25th, she was quite well.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. v, p. 118.)

ON

THE USE OF ATROPINE IN CHOLERA.

BY

T. LAUDER BRUNTON, M.D.,

F.R.C.P., F.R.S., LL.D. HON. ABER.

Received April 26th—Read June 13th, 1893.

I HAVE to ask the indulgence of the Society for bringing before it a paper founded upon only one case. My excuse for doing so is that the case is one for which I have been obliged to wait nearly twenty years, and the probability of a return of cholera during the present year is so great that it is well to discuss all means of treatment which are likely to be useful in the disease before it actually begins to spread again, either in this country or on the Continent.

In 1873, in a paper read before the British Association at Bradford, I pointed out the great likeness which exists between the symptoms of cholera and those produced by poisoning with muscarine; and I then suggested that as atropine completely antagonised the action of muscarine, and prevented death from what would otherwise have been a fatal dose, it might possibly be useful in the treatment of cholera. At that time the pathology of infective diseases was not understood as it is now. One could not point to any organism as the cause of cholera, nor did our knowledge allow of any distinction being drawn between pathogenic microbes and the poisons which they produce. I did not, therefore, attempt to discuss more closely the nature of

the poison. While the infective nature of cholera and its transmission from place to place seemed to point to a microbe as its cause, yet Lewis and Cunningham,[1] in a paper published in the year subsequent to mine, announced that they had failed to find any microbe in the blood or tissues, but had succeeded in producing symptoms of gastro-enteric irritation by the injection of boiled cholera dejecta into the blood of animals. These observations of Lewis and Cunningham appeared to show that the cholera dejecta contained the poisonous substance quite apart from any living organism which might be present in them. Since that time the researches of Koch have shown that the so-called comma bacillus is the organism most frequently found in cases of cholera, but according to Cunningham[2] other varieties of bacillus may produce symptoms indistinguishable from those of cases where the true comma bacillus is found. We may, therefore, regard it as certain that a micro-organism is the cause of the disease, although it is quite possible that this may not always be of the same character; and it is unnecessary to discuss this subject further at present, inasmuch as the subject we have to deal with to-night is not so much the effect of drugs upon the organism which gives rise to the disease as the treatment of the symptoms which the patient presents.

It is now, I think, generally agreed that these symptoms are not caused by bacilli themselves which remain in the intestine, and do not pass into the tissues, but are rather due to some poison which these bacilli produce, and which, after absorption into the circulation, acts upon various parts of the organism. When we come to inquire into the nature of this poison we find that opinions are divided about it as well as about the bacillus which generates it. According to Hueppe it is a substance resembling peptone [illegible] sical and chemical reactions, but dif[illegible] respect, that its activity

[illegible]

1 "N[illegible]," Calcutta, 1874.

2 D.[illegible]holera," by A. Crombie, M.D., [illegible]

is destroyed by boiling. It is evident from this statement that the substance regarded by Hueppe as the poison is not the same as that with which Lewis and Cunningham were dealing in their experiments with fresh cholera dejecta. I am therefore at the present moment, notwithstanding all the work which has been done upon cholera, unable to speak much more definitely about the poison than I could twenty years ago, and I may therefore fairly quote from my paper read at the British Association at Bradford, and say, "The cause of cholera is now generally admitted to be a poison of some sort, which can be conveyed about from place to place and transmitted from one person to another, through the medium of the evacuations, which either get into water and are drunk, or become dry and are taken into the mouth and nostrils in the shape of dust. Some even yet are inclined to hold that cholera results rather from peculiar atmospheric and other conditions than from the presence of a specific poison; but the fact that the disease may be conveyed from one infected locality to numerous others by a single individual, breaking out where he has stopped, and passing over those places which he has only travelled through, although those may present apparently identical conditions of air, sea, and water, shows conclusively that an outbreak of the pestilence cannot be due to these latter circumstances alone. Nor will the mere presence of the poison always produce cholera, for those who are exposed to contagion do not all become affected, and even those who have swallowed cholera stools, in which the poison is supposed to be present in its most concentrated form, have sometimes escaped with impunity. It would appear that two conditions are required, viz. the presence of the poison and the existence of a proper soil for its development. In other words, it would seem that the poison does not produce its usual effects even when it has entered the system, unless the blood and tissues are in such a state that it may act upon them. The nature of this state we cannot exactly define, but its

presence seems to be due in great measure to those conditions of atmosphere and soil which some assert to be the immediate cause of the disease, but which in reality only predispose to it.

"Without entering into this question at any greater length, I shall assume that cholera is caused by a specific poison acting upon an organism which has become in some way or other susceptible to its influence. The effects of the poison upon the body may be summed up in few words. It produces irritability of the digestive canal, immoderate secretion from the intestines, and lessened circulation both through the lungs and the body. Bearing in mind these actions, it is perfectly easy for anyone to deduce from them all the symptoms which are observed in the state of cholera collapse. From the irritability of the stomach and intestines there is constant vomiting and purging. The secretion from them is so profuse that the whole intestinal canal is speedily washed clean out; the stools are no longer feculent nor even tinged with bile, but consist of the secretion alone, pure and unmixed, and resembling rice water in appearance. The blood is thus drained of its fluid parts, and the consequence of this is intense thirst, which adds greatly to the sufferings of the patient. The blood itself, instead of coursing rapidly through the vessels as it does in health, stagnates in the great veins of the thorax and abdomen; the left side of the heart, instead of receiving from the lungs a full supply of well-aërated blood which it would propel through every part of the body, receives only a scanty driblet which leaves it almost collapsed; the arteries which proceed to the body are so empty that when they are cut across hardly a drop of blood flows from them; and even when a tube is passed through the carotid artery and aorta right up to the sigmoid valves of the heart, as was done by Dieffenbach, no blood can be drawn from it. The [illegible] blood from the interior of the body, [illegible] the vessels near the surface, [illegible], warmth, and rosy

hue of health, stagnates in the abdominal veins, and leaves the skin shrunken, pale, and cold; while that in the interior of the body, being no longer cooled by circulation near the surface, becomes hotter and hotter, till the internal temperature of the unfortunate patient is higher than it usually is in high fever, though his skin and breath are cold as ice. The blood which fills the small cutaneous veins, being no longer driven forward by fresh supplies from the arteries, becomes completely deoxidised and black, imparting to the surface a livid hue. So dark does the blood become that it assumes the colour of bilberry juice, and the colouring matter leaves the corpuscles and tinges the serum. It still retains its power to take up oxygen and give off carbonic acid, but notwithstanding this it passes so slowly through the pulmonary vessels that only about one third of the usual quantity of carbonic acid is given off from the lungs, and little oxygen being taken in, there is a distressing feeling of want of breath. The voice at the same time is hoarse, low, and weak, but this seems to be simply a consequence of the general exhaustion of the patient.

"Such are the symptoms of cholera, all rising from disturbance of the circulation and excessive intestinal secretion. The remedy we seek must, therefore, be one which has the power of removing these conditions. It may be thought that the only way to do this is to eliminate from the body the poison which is producing these results, and that so long as it is still circulating in the blood any remedy which is simply intended to counteract its effects will be administered in vain. But the researches of Fraser and others on antagonism have shown us that the elimination of a poison is not required in order to prevent its injurious or fatal action, for the administration of an antidote will deprive it of its hurtful power, and as it is with other poisons, so may it be with cholera."

If we compare these symptoms with those of poisoning by muscarine, as described by Schmiedeberg, we find a remarkable resemblance between them. They are profuse secre-

tion of tears and saliva, very violent retching and vomiting, and diarrhœa with much griping. There is contraction of the pupil, very frequent and laboured breathing, and in some animals a rapid, and in others a slow pulse, muscular weakness, cessation of the respiration, and death. On making some experiments as to the cause of dyspnœa, I came to the conclusion that the vessels of the lung were contracted by the drug, so that the blood could not readily pass from the right to the left side of the heart. After the administration of muscarine I saw the right side of the heart become distended, the lungs become pale, and the left ventricle become contracted. The administration of a small dose of atropine caused a complete change in these appearances. The right ventricle emptied itself, the lungs became rosy, and the left side of the heart again became filled. The effects led me to think that atropine might be a useful remedy in cholera, but I had no opportunity of testing this myself until last August, when two cases arrived in London from Hamburg and were brought to St. Bartholomew's Hospital. One of these was a man, Gustave Rosenbaum, æt. 35, who left Hamburg on August 27th, apparently quite well, but was very sick and suffered from diarrhœa during a rough passage. He landed at Harwich on the morning of August 29th, and came on to London the same morning. He seemed fairly well during the day. During the evening he was taken ill, had diarrhœa, with pains across the back and cramps down the legs, chiefly in the right. He had diarrhœa all night. The motions were loose but coloured. On the morning of the 30th he vomited, had no diarrhœa after 11 a.m., but suffered much from pain across the back, and was brought to St. Bartholomew's late in the afternoon. Having been summoned to an urgent case of pneumonia at some distance off in the country, I did not see the patient, but I heard he was to come in, and I discussed the treatment with the house physician. When admitted he had a sallow, grey look, his eyes were sunken, pupils equal, not contracted. His pulse was scarcely perceptible at the wrist, 148, very

feeble; the respiration was sighing, 56; the temperature 95·8°. The first sound was distinct at the apex, the second sound was not heard, and both sounds were very feeble at the base. The arms were cold, and the fingers were cold and dusky. He was ordered Liquor Hydrarg. Perchlor. half a drachm, spt. of chloroform 5 minims, and water up to an ounce every four hours, to act as an intestinal disinfectant. At ten o'clock he was very restless, complained of great pain in his back and the calves of his legs, and was ordered the mixture every two hours. At 1.20 a.m. on the 31st he was still very restless; his pulse was perceptible at the wrist, 160 per minute; the respirations were 40. The bowels were open twice, once into the bedpan and once into the bed. The motions were hardly coloured at all. He passed a small quantity of urine, which contained a small cloud of albumen, and was acid and high-coloured. At 2.15 the bowels were open, then again at 2.45, and again at 3. At 3.30 he asked for the bedpan, and then suddenly collapsed and died. Although the motions up to the last hardly presented the typical rice-water appearance, the comma bacillus was found in large numbers by Mr. Cautley in the intestine and Dr. Klein confirmed the observation.

On post-mortem examination the mucous membrane of the intestines was rather pale, with a few reddish patches on the lower part of the small intestine. The blood in the veins of the neck and of the heart was thicker and blacker than usual. The spleen was normal, and there was only one mesenteric gland at all enlarged. The other organs were merely normal.

At the same time as this patient his daughter, Casperina R—, æt. 3, was brought into the hospital.

She was taken ill upon the 24th of August, with spots on her face and diarrhœa. From this she appeared to recover, but remained somewhat languid. On the 27th she left Hamburg. She was sick on board ship, but had little diarrhœa, and on August 29th, when she came to town, was fairly well. On the morning of the 30th she

was taken ill with frequent diarrhœa and violent vomiting. The motions were slightly coloured, and there was no pain. On admission to St. Bartholomew's on the evening of the 30th she was dusky, with pale lips and sunken eyes; the pupils were equal and of moderate size, the conjunctivæ injected. The legs and arms were cold, clammy, and dusky. The tongue was moist. The pulse was 143, feeble. The respiration was 39, sighing, the temperature 97°. The impulse of the heart was feeble, the first sound absent at the apex and the second at the base.

The child was ordered milk, soda water and brandy, Liq. Hydrarg. Perchlor. ɱxv, Spt. Chloroform. ɱij, and water up to half an ounce every two hours. At eleven o'clock the child was not so well as on admission. The bowels had been open three times, and the motions were of a pale brown colour, considerable in quantity, and contained no solid matter. At 1.30 the extremities were cold, lips very pale, pulse 152, easily counted; resp. 24, long-drawn and sighing; no more diarrhœa. On August 31st I saw the child at 9 a.m.; the pulse was better, hands warmer, no diarrhœa. At 1 p.m. the pulse was feebler, the eyes were sunken and glassy, no urine had been passed. The lips were not so blue; the respiration was 20, not so sighing; pulse 124. At 5.30 the child was in the same condition, but the hands were colder; the pulse was 140. One two-hundredth of a grain of atropine was then given subcutaneously. In ten minutes the child appeared flushed, the pulse went up to 160, the respiration to 24, and was not so sighing. For a short time after the injection the child slept quietly. The bowels were not open, and no more urine was passed. There was some sickness, and in consequence of this the Liq. Hydrarg. Perchlor. was stopped, and Bism. Subnit. was given instead. Just before midnight there was more vomiting and sighing respiration, and a quarter of a grain of calomel was ordered every two hours. At 3.45 the respirations had sunk to 12, and they were long-drawn and sighing. The pulse was 140 and the surface very cold. One four-

hundredth of a grain of atropine was then injected subcutaneously. In a quarter of an hour the hands became rather warmer, the pulse remained at 140, but the respirations rose to 18 per minute. Small enemeta of water, half an ounce, were also given. During September 1st the condition varied a good deal, but the respiration did not again sink so low as before, although towards evening it fell to 15. There was considerable sickness, but only one motion in the early morning. On the 2nd the child was in the same condition, passed a greenish-coloured motion with some solid matter in the early morning. From this time the child steadily improved until she left the hospital on September 8th, completely recovered.

In this case the injection of atropine acted as a powerful stimulant when the respiration appeared about to fail, and increased the rate of breathing and the warmth of the surface. It did not appear to have any influence upon the retching, vomiting, or diarrhœa. This is precisely what one would expect from the experiments which Dr. Pye-Smith and I conducted a number of years ago, and which we reported to the British Association in 1874, 1875, and 1876. In the first series of these experiments Dr. West was associated with us in the committee of the Association. The whole of these papers were reprinted, and the pathology and treatment of cholera in general were discussed by Dr. Pye-Smith and myself in the 'Practitioner' for November, 1884, and in subsequent numbers.

In summing up our conclusions regarding treatment, we observed that two desiderata in the treatment of cholera were (1) an antiseptic to destroy the cholera bacillus and prevent it from forming poisons in the intestine, and (2) a remedy which would antagonise the action of the poison after its absorption from the intestine.

In the treatment of the case which I have described, the perchloride of mercury and calomel were used for the purpose of disinfecting the intestine, the atropine was given for the purpose of antagonising the action of the

cholera poison upon the circulation and respiration. I have already mentioned that we do not yet know the exact nature of the cholera poison; and it seems not at all unlikely that there are more than one, because, as I have already pointed out, Hueppe and Cunningham appear to have had different poisons, both of which they had obtained from cholera dejecta or cultivations from them, and M. Villiers obtained from the intestinal contents of cholera patients an alkaloid which had a marked action upon the heart, sometimes slowing it greatly, and at other times greatly increasing its rapidity. Claude Bernard has stated that in some cholera patients slowing of the heart was observable for some days before the attack, and this rather points to the presence in the circulation of a poison having an action like muscarine. It is a well-known fact that some mushrooms are intensely poisonous, while others are not, but occasionally some species become poisonous when grown under certain conditions. It seems not impossible, therefore, that the cholera bacilli, even if we suppose them to be originally of the same nature, may not only undergo variations in their appearance, but may form poisons according to the conditions which they meet in the intestine. Amongst these it is by no means improbable that muscarine itself may actually be formed, inasmuch as Brieger has shown it to be not an uncommon product of albuminous decomposition during putrefaction; and, if so, atropine is likely to be useful in certain cases. But atropine cannot be looked upon at all as a universal remedy for cholera. It is not likely to affect the profuse intestinal discharge, and will probably, therefore, do but little good in cases where this is very profuse. But in such cases as those two which I have described, where the intestinal discharge was scanty and the vascular symptoms were pronounced, atropine may be useful, and undoubtedly in the case of the little girl, Casperina Rosenbaum, a marked improvement appeared to follow immediately upon its injection, although a relapse occurred subsequently. Before concluding I may mention some interesting obser-

vations of Alt, who has found that not only is the venom of snakes excreted from the stomach when injected subcutaneously, but that toxalbumins of cholera are excreted in a similar manner, and that washing out the stomach may also be useful as an adjunct to other treatment. In a paper that I wrote in the 'British Medical Journal' I pointed out the probable utility of washing the stomach out in case of a snake-bite, but Alt's observations were only published a month or two ago, or I should have tried the effect of washing out the stomach in the case of Casperina Rosenbaum, either with water, dilute saline solution, or with a weak solution of permanganate of potash.

The general treatment of cholera is, however, too wide a question to go into here, and I must be content with simply pointing out the possible use of atropine as a cardiac and respiratory stimulant in some cases of the disease.

In conclusion, I desire to acknowledge the careful attention of Dr. Batten, house physician, and to thank him for his laborious observation of the cases.

St. Bartholomew's Hospital (August 30th, 1892).—Casperina R—, æt. 3.

History.—Child taken ill six days ago, had spots on her face, had diarrhœa, after medicine got better. Left Hamburg on Saturday, August 27th, three days ago. Somewhat languid after the diarrhœa. One hundred and eleven on board ship. No diarrhœa to speak of. Fairly well on August 29th, when she came to town. Yesterday evening child slept fairly well. This morning child first taken ill. Had frequent diarrhœa, motions slightly coloured. Child vomited after everything given except Mellin's food; no pain.

Family history.—Mother well. Father choleraic symptoms. Only child. *Past illness.*—Always healthy.

Present condition.—Dusky, eyes sunken, lips pale, tongue moist. *Pulse* 143, feeble, easily countable at the wrist. *Respiration* sighing, 39, long-drawn inspirations

at times. *Temp.* 97. *Eyes* very much sunken, pupils equal, moderate size, conjunctiva injected. *Arms:* hands cold, clammy, dusky colour. *Chest:* breath-sounds natural. *Heart:* impulse feeble, absence of first sound at apex and second sound at base. *Legs* cold, clammy, blue. *Urine.* *Abdomen* resonant, no tenderness; liver not enlarged.

11.10.—Child not so well as on admission. Bowels opened three times since admission; motions pale brown colour, no solid matter, a good deal in quantity. *No vomiting.* Ordered milk, soda water and brandy, and Liq. Hydrarg. Perchlor., ♏xv; Spt. Chloroform., ♏ij; Aq. ad ½ oz., 2dis hor.

1.30.—Extremities cold, pulse easily countable at wrist, 152. *Resp.* 24, sighing, long-drawn; no more diarrhœa; lips very pale.

August 31st, 9 a.m.—Pulse better, hands warmer, no diarrhœa.

1 p.m.—Pulse not so good as the morning. Eyes sunken. Child taking not so well. No urine passed. Lips not blue. Eyes glassy. Resp. 20, not so sighing. Pulse 124.

5.30.—Child remains in same condition. Hands colder. About 1.30 passed some urine; no motion. Pulse 140. Child will not take readily; injection of $\frac{1}{200}$ gr. of atropine subcutaneously.

5.40.—Child appears flushed. Pulse 160. Resp. 24, not so sighing.

7.30.—The last dose of Hydrarg. Perchlor. was given at 4.30. It was not repeated at 6.30 on account of the sickness. Ordered Bismuth. Subnit., gr. iss; Sod. Bicarb., gr. iiss; Spt. Chlorof., ♏iiss; Aq. Arni, ʒij p. r. n. for the sickness. Child vomited about 1 oz. Pulse 132. Resp. 20. Not so flushed. Child apparently slept quietly for a short time after the injection. Hands fairly warm. Bowels not opened. No more urine passed.

10.15.—Child vomited again. Pulse 140, resp. 24.

September 1st, 12.15.—Child vomited again at 11.45.

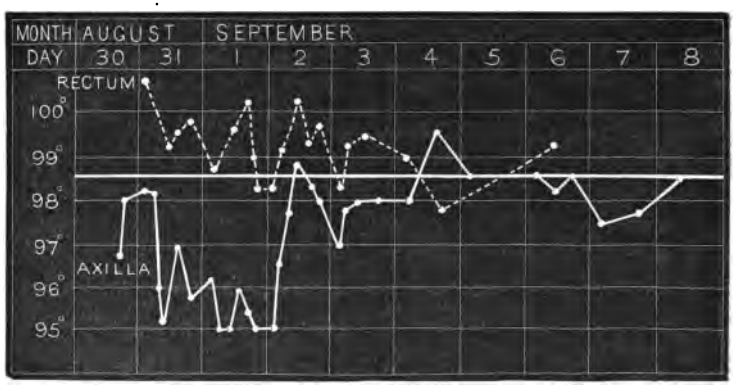
Pulse 120, respiration sighing. Ordered calomelanos gr. ¼ secundis horis.

1.20.—Not vomited again, hands rather colder. Pulse not so good, 120.

3.45.—Child vomited again at 3.15; about 5 oz. of milk given. Resp. 12, long-drawn; pulse 140. Injection of $\frac{1}{400}$ gr. of atropine.

4.—Hands rather warmer. Pulse 140, resp. 18. Enemata of ½ oz. of water.

September 1st, 9.50.—During the past six hours the child has taken only 6 dr. of milk and soda water; the enema given at four o'clock was retained as well as the one given



an hour later. The one given at 6.15 was returned with loose light-coloured motion. The child seemed brighter this morning. Pulse not so good, 120, fairly regular; hands cold, feet warm. Respiration more sighing for last hour, 20. No vomiting since 3.15.

12.45.—Vomited twice since 9.50. Child seems brighter. Pulse 140.

5 p.m.—Pulse much better, 108, fair volume; resp. 20. No vomiting since 12.30. Taken 5 oz. of milk and soda water, 1½ dr. of brandy, two enemata of ½ oz. each. Passed over 1 oz. of urine at 10.50; urine acid, very faint trace of albumen.

9.30.—Pulse not quite so good, 120. Child vomited

once this evening and retched for some time afterwards. The child slept afterwards. Taken Oss. in last twelve hours. Child rather more restless. Had arrowroot water this evening, vomited this evening after it.

11.40.—Child sleeping quietly for last two hours. Eyes shut, breathing regularly. Took 1 dr. of milk and brandy, tendency to vomit. Pulse 120, resp. 15; respiration more sighing.

2nd, 1.20.—Child in same condition. Pulse 116, regular, fair volume. Dark greenish-coloured motion passed, not very offensive. Some solid matter.

10.30.—Child very much better, eyes less sunken, seems in pain at times, no vomiting since 1.35 (vomited immediately after milk). Pulse 120, respiration 28, not so sighing in character. Urine passed into bed. Bowels open very slightly.

7.25.—Taken very well to-day, nearly 1 pint. Child much better. Bowels open once. Motion light, unhealthy-looking. Pulse 120, resp. 28. No vomiting.

3rd.—Very much improved, no vomiting, no diarrhœa. Pulse 116, resp. 28; eyes much less sunken.

4th.—Child much better; has taken bread and butter, custard pudding, and bread and milk, slept well. Pulse 120, regular. Bowels open once during the night.

5th.—Pulse 112. Child quite well, taken chicken broth, sat up in bed. Bowels not open. Temperature normal.

6th.—Child quite well, bowels not open. Ordered dose of Ol. Ricini. Urine acid, 1012, no albumen.

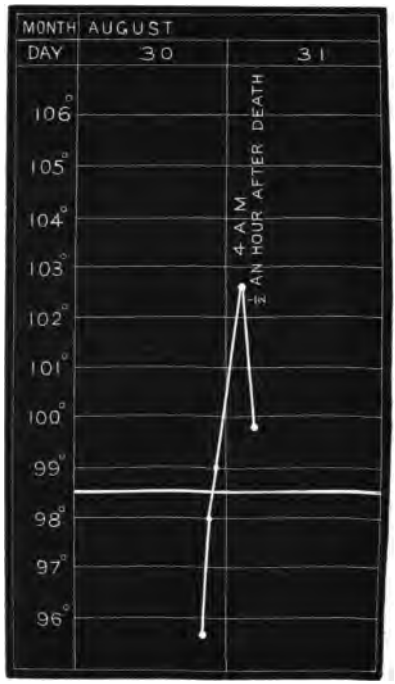
8th.—Child quite well. Ol. Ricini acted well. Child going home.

Gustave R—, æt. 35.

History.—August 30th.—Left Hamburg on Saturday, August 27th, quite well when starting, rough passage, very sick during the voyage, had diarrhœa, landed at Harwich on Monday morning, August 29th, came to town same morning, seemed fairly well during the day. In

evening taken ill, had diarrhœa, pain across the back and cramps down the legs, most in right. Diarrhœa continued all night, motions coloured, loose. This morning vomited after medicine given by doctor, no diarrhœa since 11 a.m. this morning. Great pain across the back.

Family history.—Child taken ill while at Hamburg, got well again, taken ill again last night.



Present condition.—Sallow, grey look, eyes sunken, pupils equal, not contracted, pulse scarcely perceptible at wrist, 148, very feeble. *Resp.*—Sighing, 56. *Temp.*—95·8°. *Lips* dry, very poor colour; tongue moist, not furred. *Eyes* very much sunken. Cheeks hollow. *Chest* thickly covered with hair, percussion good all over. Breath sounds natural. *Heart.*—Apex-beat distinctly felt in fifth interspace. *Sounds.*—Distinct first sound at

apex, second sound not heard; sounds very feeble at base. *Abdomen* not retracted, resonant all over, except hepatic dulness. *Bladder* not distended above pubes. No tenderness on palpation, not retracted, warm. *Arms* cold, fingers cold, dusky. *Legs* fairly warm, feet rather cold, knee-jerks not obtained. Urine passed into bed. Was ordered Liq. Hydrarg. Perchlor. ʒss, Spt. Chlorof. ɱv, Aq. ad 1 oz. 4tis hr.

10.15.—Very restless, complains of much pain in his back, and some in calves of his legs. Ordered mixture every two hours.

31st, 1.20 a.m.—Still very restless, pulse perceptible at wrist. Passed about 4 oz. of urine, B. O. twice, once into bedpan, once into bed. Taken 23 oz. milk, 1 oz. brandy. Hands cold, less pain in back, motions watery, hardly coloured. Pulse 160, resp. 40.

3.30, patient suddenly collapsed. Bowels open just at 2.15, and then again at 2.45, and again at 3 o'clock, and immediately before collapse asked for bedpan. Had taken medicine at 3, seemed no worse, very restless. Motion light brown colour, no solid matter. Urine high-coloured, fair cloud of albumen, acid.

Post-mortem made nine to ten hours after death.

External appearances.—A medium-sized man. Body moderately well nourished. Abdomen neither retracted nor swollen. Face and eyes sunken. Marked rigor mortis. The blood in the veins of the neck and in the heart was thicker and blacker than usual.

The examination of the head and neck was not allowed. *Lungs* normal, light and spongy in texture, not engorged. *Heart* normal, soft clot in the cavities, weight 10 oz. *Abdomen. Peritoneum.*—No peritonitis. External surface of the intestines rather dulled in appearance, and thought by some of those present to be soapy to the touch. General colour rather pale than otherwise, but vessels injected somewhat. The same external appearance throughout all the length of intestine. *Stomach.*—Normal, contained some bilious fluid. *Intestines.*—In

upper part of small intestine yellow fluid like the bile-coloured stuff usually found here. Lower down the fluid became paler and thicker (pale greenish yellow), but it was at no point colourless nor "rice-water" in character. The mucous membrane was rather pale in colour with a few reddened patches in lower part of small intestine, but otherwise looked normal. *Liver.*—Rather small, otherwise normal, weight 44 oz. *Spleen.*—Small, weight 5 oz., section normal. *Abdominal lymphatics.*—Only one gland was at all enlarged, and that not greatly, viz. a mesenteric gland near the valve. *Kidneys.*—Normal in appearance though small, weight 10 oz. the pair.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 122.)

A CASE

OF

MALIGNANT DISEASE OF THE THYROID GLAND,

WITH MOST UNUSUAL COURSE.

BY

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Received February 13th—Read June 13th, 1893.

On the 13th of January, 1891, I was consulted by Miss L—, æt. 52, sent to me by Dr. Wright of Huddersfield with the following history:

Miss L— had consulted Dr. Wright about six weeks previously, complaining of difficulty in respiration with much cough and expectoration; she had brought up a mouthful of blood for the first time the day previous to her visit. On examination Dr. Wright found a good deal of bronchial catarrh; externally a swelling at the right side of the thyroid was seen, which seemed solid and fixed. During the time of his attendance no appreciable altera-

tion in its size took place, and under treatment everything settled down except the difficulty in respiration, which he believed was entirely due to pressure caused by the enlargement. Internal and local iodine treatment having failed to reduce the latter, Dr. Wright felt that nothing short of the removal of the right lobe of the thyroid was likely to be of any benefit.

When Miss L— consulted me on the 13th January the external conditions entirely corresponded to Dr. Wright's description. There was a very hard but not very large tumour about the size of a Tangerine orange to be felt in the region of the right lobe of the thyroid, rather fixed to the larynx, but the skin above it was moveable. It caused no pain and no inconvenience in swallowing, and in fact she was hardly aware of its existence. Her voice was perfectly normal and clear, but occasionally, especially after speaking a little, the inspiration was very stridulous.

On laryngoscopic examination I was not a little startled to see at the first glance below the vocal cords, and much more developed towards the left side—that is to say, to the side *opposite* the external tumour—a large whitish ulcerating swelling, which on its extremity on the left seemed to nearly reach up to the level of the left vocal cord. It was completely covered by the vocal cords on phonation, and the mobility of the cords was perfectly unimpaired. When, however, the patient inspired deeply one saw that this growth to a very great extent filled the subglottic cavity, and the dyspnœa thus was certainly only in part due to the pressure of the external tumour, the greater share being taken by the internal narrowing. When I looked with the laryngoscope for the second time the tumour without any apparent cause actually began to bleed, though the hæmorrhage was only insignificant.

There could not be the least doubt in my mind after this that one had to do with a malignant growth, the question being whether it had started from the subglottic cavity, and on the one hand extended upwards to the left, on the other broken th[illegible] ico-thyroid membrane

on the right and invaded the right lobe of the thyroid gland; or whether the whole affection had started from the thyroid gland itself, and the invasion of the subglottic part of the larynx was only a secondary phenomenon.

This question of course could only be elucidated at a later period by microscopic examination of the growth itself. Therapeutically there could, in my opinion, seeing the large extent of the growth, not be the least doubt that the case was not suitable for any radical operation, and that the only proper measure could consist in the performance of early tracheotomy. Mr. Butlin, who subsequently saw the patient in consultation with me, entirely shared my views. I then wrote to Dr. Wright, and advised early tracheotomy, the condition being in my opinion distinctly dangerous, inasmuch as the least access of acute œdema, which so often supervenes in cases of this sort, would in all probability have been directly fatal by complete occlusion of the trachea. The patient returned to Huddersfield, and Dr. Wright having approved of my proposition, came again up to town on the 16th January.

On the following morning I performed low tracheotomy, assisted by Messrs. Miller and Carstairs of St. Thomas's Hospital, Mr. Tyrrell giving chloroform. I of course selected the lower operation in order to be as far as possible away from the seat of the disease. The operation did not offer the least difficulty, but when the trachea was laid bare it was seen that it was already to some extent pushed towards the left, whilst at the same time it was found that the right carotid had been displaced backwards and outwards, and was very distinctly felt pulsating just behind the external tumour.

The patient at first made an excellent recovery, and the temperature never rose during the first week, but already at that time two very uncommon symptoms made their appearance. In the first place the patient brought up two or three times within the twenty-four hours with great difficulty, and only after much coughing and strain-

ing, a brownish-red, sometimes round, sometimes oval mass of a tenacious, leathery character, varying from the size of a hazel-nut to that of a small cherry, altogether unlike any bronchial secretion I have ever previously seen. These big lumps usually obstructed before being expelled the opening of the tube, and caused for the time being considerable dyspnœa; as a rule they could only be removed with great difficulty. In the free intervals the breathing was absolutely quiet, and there was not the least symptom of general bronchitis or of localised pneumonia, the temperature remaining normal all the time. The attacks of coughing, however, which preceded the expulsion of these lumps, always exhausted the patient very much.

The second phenomenon was equally curious. The patient's voice, which, previous to the operation, had been perfectly normal, after the operation varied in the most extraordinary manner: sometimes it was just as normal as previous to the operation; at other times it was not only absolutely aphonic, but the patient could not produce any sound at all, and one had certainly the impression that on attempting to speak she was seized with a spasm of the glottis. On laryngoscopic examination no clue was detected of the cause of this curious phenomenon, both cords being perfectly white, and moving with complete freedom.

Whilst with the exception of these two symptoms the recovery and the healing of the wound made very satisfactory progress, suddenly on Saturday, the 24th, *i. e.* a full week after the operation, an unfavorable change occurred. The patient had from the Friday to the Saturday a bad night, owing to much coughing, and when I saw her on Saturday morning was much exhausted. Careful examination failed to elicit any definite change. When I came, however, in the evening of the 25th I found her very feverish, the temperature without any rigor having gone up to 101·4°, the pulse to 120, and at the same time very considerable cardiac disturbance had occurred; the heart's action was

absolutely irregular, a series of quick beats would be suddenly followed by a shorter or lesser complete stop; then there would be sometimes many, sometimes few quick beats, again a pause, again one or two beats, again a pause, a series of beats, &c. In a word no type in the irregularity could be detected. In the afternoon of the same day also a sort of general breathlessness had made its appearance, totally different from the previous choking attacks due to the obstruction caused by the large pellets of secretion. There was no lividity, and the breathing was by no means unduly hurried, the respirations being about twenty-four a minute.

Considering these curious phenomena, I came to the conclusion that all of them were probably to be attributed to one and the same cause, viz. to irritation of the right pneumogastric nerve in consequence of pressure of the growth; the general breathlessness, rise of temperature, and the curious expectoration being due to irritation of the pulmonary branches, the change in the voice to irritation of the laryngeal, and the irregularity of the heart's action to irritation of the cardiac branches. I gave the patient digitalis, acetate of iron, and tincture of valerian. On the morning of the 26th the temperature had fallen to 98·8°, but the same night it again rose to 100·8°, and in the course of the 27th it gradually went up to 102·5°. On the 28th in the morning it fell to 99·4°, and in the evening to the normal; on the afternoon of that day I had a consultation with Dr. Broadbent, who carefully examined the patient and did not find any definite lesion anywhere. He entirely shared my opinion that all phenomena were due to irritation of the pneumogastric nerve. He also approved of the treatment pursued.

Within the next week a steady and pleasing improvement took place, and the temperature remained normal. The cardiac irregularity slowly but steadily got better under the influence of digitalis, though a very distinct systolic murmur equally slowly developed, which certainly had not been present previous to the cardiac attack.

The voice also gradually got better, and the patient, as a rule, when closing the opening of the tube with her finger could speak in her normal voice, though occasionally still the attacks of spasm of the glottis combined with aphonia were present. The only thing which did not show much change was the expectoration. Still twice or three times daily the brownish-red lumps were expectorated, but even those on the whole were less tenacious than formerly, and were expelled with less difficulty. Once or twice the patient had an attack of general breathlessness, but not so intense as on the first occasions. On Wednesday, the 4th of February, she for the first time drove out a little, and this was repeated on the following days. Her general health meanwhile considerably improved. On Friday Dr. Wright came to town, and a consultation having taken place between him and me on Saturday, the patient on the same day left under his charge for Huddersfield, wearing a rectangular (Durham's) silver tracheotomy tube.

During the first part of the year I occasionally heard from Dr. Wright that the patient was very slowly but steadily losing ground without any new symptoms having made their appearance. From July till the end of November I heard nothing more, and had already intended to write to Dr. Wright and ask him for further particulars about the case, which I had little doubt would have ended fatally before that time, when to my great surprise I received in the last days of November a letter from the patient herself, asking for an appointment on the 27th of that month, and which letter was followed by another from Dr. Wright, in which he informed me that he was much interested to know my opinion of her case. The disease during the summer months had seemed somewhat in abeyance. There had been no hæmorrhage and very much less expectoration than formerly. The general emaciation, however, had seemed to steadily continue, and the general outlook seemed dark.

When the patient entered my room on the 27th of

November I was first of all struck with the very considerable emaciation which had taken place since I had last seen her in February; and secondly, with the absolute clearness and natural character of her voice—a circumstance which seemed the more striking, as already in February the intra-tracheal growth had apparently reached up to nearly the level of the left vocal cord. On inquiries I learned that there had been absolutely no pain, no dysphagia, no difficulty in breathing; the breath had never been fœtid, there had been no expectoration of more solid particles, and that the only but great trouble was the constant wearing of the tube, which, according to the patient's statements, made her life a misery. Expectoration through it she stated to be exceedingly difficult and tiresome, and according to her assertion her life was mostly spent in removing and putting in the internal tube.

On external examination it was seen that the emaciation of the face extended to the body in general; the tumour on the right side of the neck was considerably larger than it had been in February, and was now the size of an average apple. It was *exceedingly* hard, slightly moveable in the direction from behind forward, and *vice versâ*, but not in the direction from above downward, and was apparently continuous with the trachea. It was not tender on pressure, the skin over it could be freely moved, and there were no enlarged glands in its neighbourhood, or indeed anywhere in the neck to be felt. Condition of the lungs and heart normal.

On laryngoscopic examination I hardly trusted my eyes when I saw that the subglottic tumour which Mr. Butlin, Dr. Wright, and I had so clearly seen (I myself on a good many occasions) had *completely disappeared*, and that it was now possible to see a long way down into the trachea. There, corresponding to the external tumour on the right side, a very considerable inward bulging was seen on the right side, a smaller one bulging forward the posterior wall, and again at the same level another

smaller one slightly bulging in the left lateral wall of the tube. On the border of the right lateral and posterior swellings two irregular little knobs could be seen; otherwise the mucous membrane covering these bulgings appeared to be tolerably smooth, and there was at any rate no evidence of deep ulceration. Whether these projections represented mere inward bulgings of the walls of the trachea, or actual intra-tracheal growths, it was impossible to decide.

From the observations of the patient herself and of a sister of hers who accompanied her I made out that the correctness of the diagnosis arrived at at the beginning of the year was more than doubted by them, and that the family had become almost convinced that the tumour was of a benign nature and could be removed, thereby abolishing the pressure upon the windpipe, and enabling the patient to dispense with the tracheotomy tube, the latter object being apparently the one on which all her thoughts were concentrated. I at once wrote to Dr. Wright, and whilst fully admitting the very unusual features of the case, such as the surprisingly long duration of life, the absence of pain, cachexia, dysphagia, enlargement of glands in the neck, and—above all—the disappearance of the intra-tracheal growth, which ten months previously had formed the most prominent feature of the case; nevertheless considering the history—the progressive emaciation of the patient, the increase in size of the external tumour, and the undoubted fact that at one period of the illness there had been an ulcerating tumour in the trachea—I could not alter my original opinion.

After the result of my [illegible] I had spontaneously offered the [illegible] with Mr. Butlin. The patien[t] [illegible] to be anxious to have p[ost] [illegible] independent opinion, a[nd] [illegible] consultant. [illegible] only that the [illegible]

case, because the present appearances alone without the aid of such history would hardly enable anybody to come to a definite opinion. Dr. Macdonald saw Miss L— on the 28th, and from the result of his examination and from what the patient told him (the description, as we afterwards found, was a *very incomplete* one indeed) very naturally came to the conclusion that the tumour in all probability was merely a hard goitre pressing upon the trachea, and that an attempt could well be made to remove it.

On the following day a consultation took place between Mr. Butlin, Dr. Greville Macdonald, and myself. Mr. Butlin's view was almost identical with my own. He stated that he had never seen a benign tumour of the thyroid gland of such extreme hardness. He laid great stress upon the internal swelling on the *left* side of the trachea, and he declared that he would not undertake even an exploratory operation with a view to radical removal, because the danger of such exploratory operations was great, and he felt that it would not be possible to finish the operation.

When Dr. Macdonald heard the full previous history of the patient he stated that his opinion thereby of course became considerably modified, but still he said that if he were the patient himself he would run the risk of an exploratory operation. I held to my first opinion.

In view of this conflict of opinions we all agreed that Miss L— ought to see another surgeon, and that Sir William MacCormac should be consulted. Sir William examined the patient without knowing anything of her previous history. He too considered the goitre exceedingly hard, uncommonly so for a benign growth; he also found that whilst freely moveable in the direction from the front backwards, it was not so in an up and downward direction, and above all that it was very firmly attached to the trachea and could not be separated from this at all, and considered an exploratory operation feasible. Afterwards heard the full history of the case he

looked more gravely upon the chances of an operation than before, but still considered it justified, seeing the urgent wish of the patient and the sad prospect anyhow. He consented to make such an exploratory operation, warning Miss L— at the same time that it was impossible to foresee whether the operation could be completed at all, and plainly told her that under all circumstances it would be dangerous. Miss L— then once more asked my own opinion, when I simply told her that she must now decide for herself, that my own opinion was not shaken, but that I admitted that the case was most unusual in several respects, and that I did not wish to debar her from taking what possibly was her only chance.

Miss L— decided to undergo the operation, and on December 19th, 1891, it was performed by Sir William MacCormac, Mr. Priestley, Mr. Robinson, and Mr. De Méric assisting, and Mr. Tyrrell giving chloroform, whilst Dr. Greville Macdonald, Dr. Wright, who came on purpose from Huddersfield, and I were present.

The first part of the operation presented no difficulty at all, and indeed the tumour on all its aspects except the tracheal shelled out so easily from the surrounding structures, and looked so entirely like a common fibrous goitre, that all the time I could not help asking myself whether it was after all possible that the tumour was really only an uncommonly hard but otherwise innocent goitre, and whether Mr. Butlin and I might not have been deceived in what we originally had seen. However, when the operation had arrived at that stage that the tumour was merely to have been separated from the trachea, this was found to be impossible, as they were inseparably connected with, and indeed contiguous to, one another. Sir William had therefore to be satisfied with merely removing the external portion. The knife on cutting through the tumour made a loud grating noise, and when the external portion was removed, it was found to represent a hard fibrous goitre, containing several cysts with calcareous walls within itself, and having undergone in

its central portion, where it was contiguous with the trachea, an obvious malignant degeneration, this part of the tumour being distinguished even to the naked eye by its colour and appearance from the surrounding merely hyperplastic portions. Rather free parenchymatous hæmorrhage followed the removal of the external portion, which was finally stopped by the application of Penghawar wool.

The patient recovered from the operation without any incident whatsoever. Microscopic examination of the growth made by Mr. Shattock showed that it was an encephaloid carcinoma, starting from the gland cells themselves.

About a fortnight after the operation Sir William requested me to see the patient again. He had found a sort of "moveable velum" hanging down from the upper border of the opening in the air-tube, which he thought acted as an imperfect valve obstructing the expiratory current, and he wished my opinion thereon, as the patient's urgent wish, viz. to dispense with her tube, could not be complied with so long as this obstruction existed.

I saw Miss L— on January 4th of the present year, when I found her even more emaciated than a fortnight ago, but otherwise still in—comparatively speaking—surprisingly good condition. Her voice was still perfectly clear, there was no dysphagia, no pain, and she stated that she could breathe better; a statement which was corroborated by the nurse, though it was not easy to understand how the partial removal of a tumour situated above the tracheotomy tube could have relieved the breathing, unless this had exercised some pressure upon the pneumogastric nerve, of which of late there had been no proof whatever. The external wound had not yet healed, there being a deep sinus, which was kept plugged with iodoform gauze. On removal of this it was seen that the wound was granulating freely. On examination with a good light through the tracheotomy wound I found a projection corresponding to Sir William MacCormac's description hanging down from the right lateral wall

into the trachea and down to close to the upper border of the tracheotomy wound. Its surface, however, was distinctly ulcerated—a fact of which Sir William by my wish convinced himself. It was, as he had previously found, slightly mobile, but certainly gave the probe with which I tried to move it upwards a sensation as if it possessed a good deal of weight. I introduced a small mirror into the wound, but could not look round the projection. My opinion of this projection was that it could be only one of two things, viz.—

1. An undermined, infiltrated, and ulcerating part of tracheal mucous membrane lifted from its base through the underlying ulcer, or—

2. The lower surface of a projecting part of the cancer itself. The second possibility I considered as by far the more likely one of the two.

Sir William, Mr. Priestley and I discussed the advisability or otherwise of removing this, but were unanimously against it, inasmuch as it was impossible to decide its exact nature and extent; further, as the danger of hæmorrhage from it into the trachea undoubtedly was great, especially after our experience of the external wound during Sir William's operation a fortnight previously.

This was the last occasion when I saw the patient. She returned shortly afterwards to Huddersfield. The external wound gradually closed completely, and she again recovered, I was informed by Dr. Wright, to such a surprising degree from the operation that as late as September last she travelled, during the Leeds Musical Festival, every day from Huddersfield to Leeds and back.

Shortly after this, however, the external growth again rapidly increased, breathlessness became a prominent symptom, and from time to time smart hæmorrhages occurred.

On January 9th Dr. Wright made the following note in his case-book:—"Complete aphonia, external growth much increased in size."

On January 11th "I was called up in the middle of the night, and found her suffering from a severe attack of

hæmorrhage, which ultimately yielded to ice suction and Tinctura Hamamelis, ℥xx every hour."

On the 12th "there was a return of hæmorrhage, and the pulse was intermittent."

On the 13th "in the afternoon hæmorrhage came on very violently, the tube was changed constantly, but with no relief. I could hear the flapping of something at the bottom of the tube. Patient apparently suffocating. As a last resource I introduced a celluloid tube connected with a strong suction syringe, and tried the effects of strong suction. Nothing came, but the tracheotomy tube on the withdrawal of the other became suddenly blocked, and I thought the end had come. Suddenly, with a violent expulsive effort, a piece of the growth was shot out on to the bed, and the patient, half dying, suddenly became easy. This improvement continued until the 24th, when there was a return of the hæmorrhage."

"On the 25th I saw her twice during the day, and felt sure that matters would soon reach a crisis. The breathing was laboured, and the same flapping noise could be heard. I was called to her at 2 a.m., and she died at 3.20 a.m. from suffocation produced by a not to be restrained hæmorrhage. So much for the last history. As a matter of personal interest I may mention that Miss L— showed the same marvellous fortitude and entire unselfishness up to the very last, and thanked me personally in writing quarter of an hour before her death."

Post-mortem.—"This was only a modified one, having strict relation to the affected parts. I made an incision from the chin to the middle of the sternum, cut through the cartilages of the ribs, and sawed through the sternum rather below the middle. I made an effort in removing the larynx and trachea to include the external growth, but it was intimately adherent to the skin, was of the consistence of very ripe cheese, and broke down on the slightest touch.

"The larynx and trachea being removed, I slit them up on the anterior surface, going through the old tracheotomy

wound. I then carefully examined the bronchial glands, but found no enlargement whatever. This done, I dissected the pneumogastric nerve on the right side down to its expansion at the root of the lung, and found that it was in no wise involved." So far Dr. Wright.

Of the larynx and trachea which you see before you, the following description is kindly given by Mr. Shattock.

"The thyroid gland is the seat of a malignant new formation, which extends backwards from the right side between the upper part of the trachea and the œsophagus, in which situation it passes beyond the middle line and approaches closely to the left lobe of the gland, but without being actually continuous with it; the growth as it lies on the right side of the cricoid cartilage and trachea has a vertical measurement of about 4 cm.

"Into the upper part of the trachea there projects a broadly pedunculated slightly lobular process of the tumour 4 cm. in length, and of a diameter so as to completely fill the canal: its surface is smooth and its texture extremely soft. The pedicle of the intra-tracheal process measures in the vertical direction 2 cm., and is continuous with the thyroidal growth through the right and posterior walls of the trachea. The summit or highest border of the growth within the trachea is 5 cm. below the level of the lower border of the cricoid cartilage.

"Above this there hangs into the canal, by a slender pedicle, a second smaller tumour; with its stalk this measures 1·5 cm. in length, and appears as a clubbed or pyriform process ·7 cm. in extreme breadth. Its exact site of attachment is 1 cm. below the lower edge of the posterior border of the cricoid cartilage, and it is separated by a distinct interval of normal mucous membrane from the upper limit of the pedicle of the larger growth. The posterior wall of the trachea, for a distance of 3 cm., is bulged forwards by the growth before referred to as lying between this tube and the œsophagus, and it is over this, exactly in the middle line, that the pedicle of the lesser intra-tracheal growth is attached.

"Immediately on the right of the pedicle is a ragged soft tag of tissue, somewhat larger than it, and suggesting that a third process of the growth has at some time been detached."

Remarks.—The foregoing description of what I do not hesitate to say has been the most extraordinary case in my practice speaks for itself. I cannot sufficiently thank Dr. Wright for having procured the specimen, for I strongly feel that without an actual demonstration of this my description might justly have been received with some scepticism. The total disappearance of a comparatively big ulcerating growth in the trachea, without leaving any obvious traces of its existence on the mucous membrane from which it sprang, constitutes, so far as my knowledge goes, a hitherto undescribed phenomenon in tracheal pathology, and its occurrence would have remained somewhat inexplicable if not fortunately we had found at the post-mortem the upper and smaller ulcerating growth hanging down by a thread-like pedicle from the wall of the trachea. Just as this growth, which I am able to confidently say did not exist at the time when Mr. Butlin, Dr. Greville Macdonald, and I last examined the patient laryngoscopically, would not have left the least appreciable trace of its existence if its pedicle had sloughed close to its base, nothing now can be more natural, in view of the present condition of things, than to assume that a similar growth, only larger and higher up, existed when the patient first came to see me, and that it had sloughed away when she came up again in November, 1891. Mr. Shattock's final remark directly favours this hypothesis. Under all circumstances, however, it will remain a matter of curious interest and importance that, fully ten months after the existence of an ulcerating tumour in the windpipe definitely established by three observers, justifiable doubts as to the malignant nature of the disease could have arisen in the minds of two other competent authorities. In this respect the case certainly teaches a most important lesson.

The fact that Dr. Wright at the post-mortem examination found the pneumogastric nerve in no wise affected makes, of course, the occurrence of the curious phenomena observed after the tracheotomy even more obscure. Still I am inclined to refer them, if not to actual affection, yet to irritation of that nerve, and would draw attention in that respect to the observation that at the time of the tracheotomy the right carotid had been displaced backwards and outwards, and was felt pulsating very distinctly behind the external tumour. It is but fair to assume that the right vagus, too, might have been at that time stretched and irritated.

Finally, I wish to draw attention to the remarkable fact that this originally infiltrating tumour became pedunculated, as shown by both the projections now visible in the trachea, as soon as it extended into the lumen of the tube. This appears to be not a peculiarity of this case, as I have already on a previous occasion described,[1] together with Mr. Shattock, a case of sarcoma of the thyroid, which became pedunculated on its perforating the trachea. This would seem to be an important fact, which Mr. Shattock suggests may be explained by the growth meeting with no resistance as soon as it extends into an open tube.

[1] 'Transactions of the Pathological Society,' vol. xxxix, 1888, p. 42.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. v, p. 126.)

DESCRIPTION OF PLATE VI.

A Case of Malignant Disease of the Thyroid Gland with most unusual course (Dr. FELIX SEMON).

The drawing shows the extension of the thyroid growth into the trachea, occurring in the form of two pedunculated tumours, which are separated from one another by a distinct interval of normal mucous membrane. Close to the base of the upper tumour is a rag of tissue, which probably formed the base of the pedicle of a third tumour, which sloughed away and completely disappeared during the patient's life.

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